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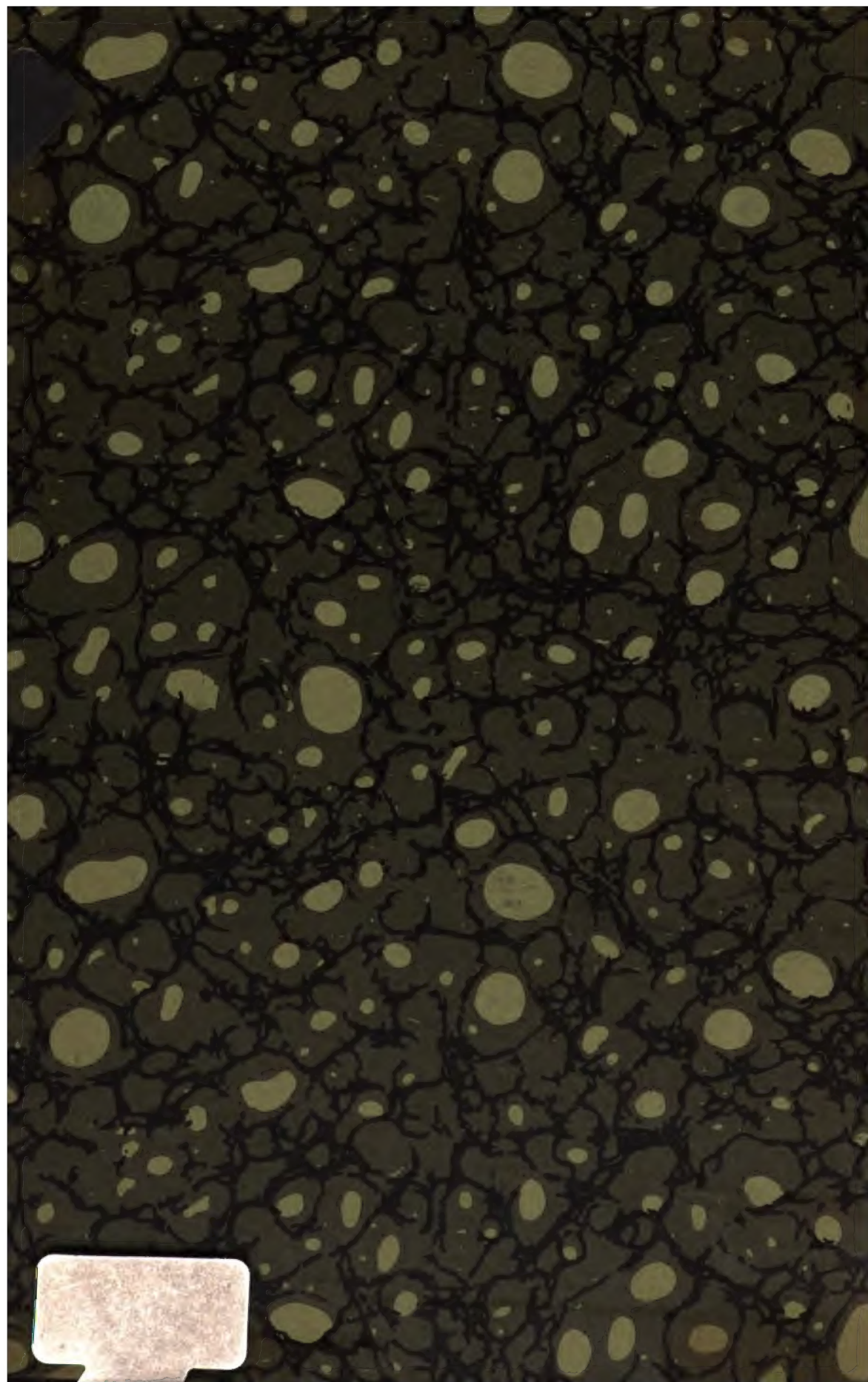
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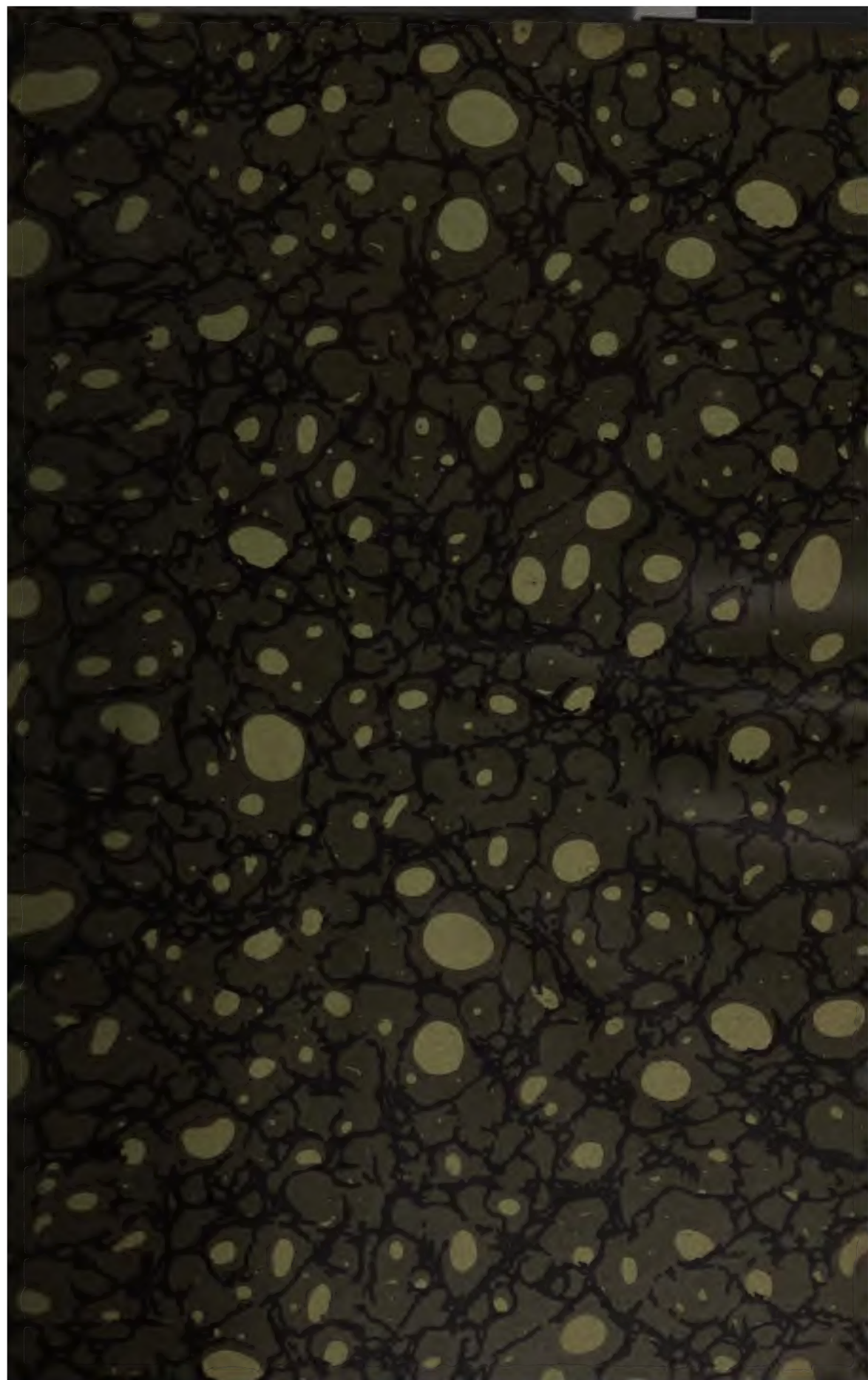
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# **SPECIAL PATHOLOGY AND THERAPEUTICS**

OF THE

## **DISEASES OF DOMESTIC ANIMALS**

BY

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## Explanation to the Abbreviations in the Reference to Literature.

A. L.	= Állatorvosi Lapok (Budapest).
Amer. V. Rev.	= American Veterinary Review (New York).
An. (Anim.) Ind.	= Annual Report of the Bureau of Animal Industry (Washington).
Ann.	= Annales de médecine vétérinaire (Bruxelles).
A. P.	= Annales de l'Institut Pasteur (Paris).
Arb. d. G. A.	= Arbeiten aus dem Kaiserlichen Gesundheits-Amte (Berlin).
A. d'Alf.	= Archives vétérinaires d'Alfort.
Arch. f. exp. Path. u. Phmk.	= Archiv für experiment. Pathologie und Pharmakologie (Leipzig).
A. f. Tk.	= Archiv für wissenschaftliche und praktische Tierheilkunde (Berlin).
Arch. f. Vet.-Wiss.	= Archiv für Veterinärwissenschaften (St. Petersburg).
A. vet.	= Arhiva veterinara (Bucarest).
Beitr. z. path. An.	= Beiträge zur pathol. Anatomie u. allgem. Pathologie (Jena).
B. kl. W.	= Berliner klinische Wochenschrift.
B. t. W.	= Berliner tierärztliche Wochenschrift.
B. Mt.	= Badische tierärztliche Mitteilungen (Karlsruhe).
Bull.	= Bulletin de la Société centr. de médecine vétérinaire (Paris).
Cbl. f. B. (Bakt.)	= Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten. Originale (Berlin).
Cbl. f. med. Wiss.	= Centralblatt für medizinische Wissenschaften (Berlin).
C. R.	= Comptes rendus des séances de l'Académie des Sciences (Paris).
Comp. Path.	= The Journal of comparative Pathology and Therapeutics (London).
Diet.	= Dictionnaire pratique de médecine et de chirurgie vétérinaire (Paris).
D. A. f. kl. M.	= Deutsches Archiv für klinische Medizin (Leipzig).
D. m. W.	= Deutsche medizinische Wochenschrift (Berlin).
D. t. W.	= Deutsche tierärztliche Wochenschrift (Hannover).
D. Z. f. Tm.	= Deutsche Zeitschrift für Tiermedizin und vergleichende Pathologie (Leipzig).
Diss. (Inaug.-Diss.)	= Inaugural-Dissertation.
Dresdn. B.	= Jahresbericht der tierärztlichen Hochschule in Dresden.
Ergebn. d. Path.	= Ergebnisse der allgemeinen Pathologie und pathol. Anatomie (Wiesbaden).
F. d. M.	= Fortschritte der Medizin (Berlin).
F. d. Vhyg.	= Fortschritte der Veterinärhygiene (Berlin).
Hb. d. p. M.	= Handbuch der pathogenen Mikroorganismen; redigiert von KOLLE & WASSERMANN (Jena).
H. (Hann.) Jhb.	= Jahresbericht der Kön. Tierarzneischule zu Hannover.
Holl. Z.	= Tijdschrift voor Veeartsenijkunde.
Hyg. de viande	= L'hygiène de la viande et du lait (Paris).
J. du Midi	= Journal des vétérinaires du Midi.
J. of comp. Path.	= The Journal of comparative Pathology and Therapeutics (London).
J. vét.	= Journal de médecine vétérinaire et de zootéchnie (Lyon).
Kongr.	= Internationaler tierärztlicher Kongress (Bern, Baden-Baden, Budapest, Haag).
Közl.	= Közlemények az összehasonlító élet-és kórtan köréből (Budapest).
Leipz. B.	= Bericht des Veterinär-Institutes zu Leipzig.
Maanedsskr.	= Maanedsskrift for Dyrlaeger (Köbenhavn).
Mag.	= Magazin für die gesamte Tierheilkunde (Berlin).
M. Orv. Arch.	= Magyar Orvosi Archivum (Budapest).
Med. vet.	= Il medico veterinario (Torino).
Mitt. d. G.-A.	= Mitteilungen des Kaiserlichen Gesundheitsamtes (Berlin).
M. (Münch.) Jhb.	= Münchener Jahresbericht (Jahresbericht der königl. Zentral-tierarzneischule in München).

# Explanation to the Abbreviations in the References to Literature. XVII

M. m. W.	= Münchener medizinische Wochenschrift.
M. t. W.	= Münchener tierärztliche Wochenschrift.
Mod. Zootatro	= Il moderno Zootatro (Torino).
Monh.	= Monatshefte für praktische Tierheilkunde (Stuttgart).
Neur. Cbl.	= Neurologisches Centralblatt (Leipzig).
N. Ere.	= Il nuovo Ercolani (Pisa).
Orv. H.	= Orvosi Hetilap (Budapest).
O. M.	= Österreichische Monatsschrift für Tierheilkunde (Wien).
O. B.	= Revue für Tierheilkunde und Tierzucht (Wien).
O. Vj.	= Österreichische Vierteljahrsschrift für Veterinärkunde (Wien).
O. Z. f. Vh.	= Österreichische Zeitschrift für wissenschaftliche Veterinärkunde (Wien).
Pr. Mt.	= Mitteilungen aus der tierärztlichen Praxis im preussischen Staate (Berlin).
Pr. Mil. Vb.	= Statistischer Veterinärsanitätsbericht über die preussische Armee (Berlin).
Pr. Vb.	= Veröffentlichungen aus den Jahresveterinärberichten der beamteten Tierärzte Preussens (Berlin).
Pr. vét.	= Le progrès vétérinaire (Alfort).
Rec.	= Recueil de médecine vétérinaire (Alfort).
Rep.	= Repertorium der Tierheilkunde (Stuttgart).
Revista	= Revista di medicina veterinaria (Bucarest).
Rev. f. Tk.	= Revue für Tierheilkunde und Tierzucht (Wien).
Rev. gén.	= Revue générale de médecine vétérinaire (Toulouse).
Rev. vét.	= Revue vétérinaire (Toulouse).
Schw. A.	= Schweizer Archiv für Tierheilkunde (Zürich).
Schw. A. f. Tz.	= Schweizer Archiv für Tierheilkunde und Tierzucht (Bern).
S. B.	= Sächsischer Bericht (Bericht über das Veterinärwesen im Königreich Sachsen).
Soc. biol.	= Comptes rendus de la Société de biologie (Paris).
The Vet.	= The Veterinarian (London).
Tidskr.	= Tidsskrift for Veterinærer (København).
Tm. R.	= Tiermedizinische Rundschau, mit besonderer Berücksichtigung der vergleichenden Pathologie (Halle).
T. R.	= Tierärztliche Rundschau (Friedenau-Berlin).
T. Z.	= Tierärztliches Zentralblatt (Wien).
Trop. Vet.	= The Journal of Tropical Veterinary Science (Calcutta).
Ung. Vb.	= Jahresbericht über das Veterinärwesen in Ungarn (Budapest).
Vet.	= Veterinarius (Budapest).
V. A.	= Virchow's Archiv für pathol. Anatomie u. Physiologie und für klin. Medizin (Berlin).
V. J. (V. Journ.)	= The Veterinary Journal (London).
Vet. Jhb.	= Jahresbericht über die Leistungen auf dem Gebiete der Veterinärmedizin (Berlin).
Vet. Rec.	= The Veterinary Record (London).
V. f. T. (Vortr. f. Tzte.)	= Vorträge für Tierärzte (Leipzig).
W. f. Tk.	= Wochenschrift für Tierheilkunde und Viehzucht (München).
Z. f. Biol.	= Zeitschrift für Biologie (München).
Z. f. Flhyg.	= Zeitschrift für Fleisch- und Milchhygiene (Berlin).
Z. f. Hyg.	= Zeitschrift für Hygiene und Infektionskrankheiten (Leipzig).
Z. f. Imm.	= Zeitschrift f. Immunitätsforschung und exper. Therapie (Jena).
Z. f. Infkr.	= Zeitschrift für Infektionskrankheiten, parasitäre Krankheiten und Hygiene der Haustiere (Berlin).
Z. f. kl. Med.	= Zeitschrift für klinische Medizin (Berlin).
Z. f. Vet.-Wiss.	= Zeitschrift für praktische Veterinär-Wissenschaften von Pütz (Bern).
Z. f. Tm.	= Zeitschrift für Tiermedizin. Neue Folge der Deutschen Zeitschrift für Tiermedizin und der Österreichischen Zeitschrift für wissenschaftliche Veterinärkunde (Jena).
Z. f. Vh.	= Zeitschrift für Veterinärkunde (Berlin).
Z. f. Phys.	= Zentralblatt für Physiologie (Leipzig u. Wien).
Zool. méd.	= Traité de zoologie médicale et agricole (Paris).





# Diseases of the Respiratory Organs

## SECTION I.

### DISEASES OF THE NASAL CAVITIES AND OF THE ACCESSORY CAVITIES OF THE NOSE

#### I. Hemorrhage from the Nose. Epistaxis.

(*Hæmorrhagia narium*, *Rhinorrhagia*.)

By hemorrhage from the nose proper is understood a hemorrhage from the vessels of the nose or from those of the accessory nasal cavities.

**Etiology.** Epistaxis is sometimes seen in otherwise perfectly healthy horses in consequence of a congenital predisposition, probably hemophilia, and may then occur without any obvious cause at all or from a quite insignificant external cause. Predisposition of this kind to epistaxis appears to be congenital, particularly in highly bred horses, and it often disappears with advancing age.

The most common causes of epistaxis are traumatic injuries to the head, such as a blow or a thrust, or injuries to the nasal mucosa, which may also be due to the introduction of the laryngoscope, to convulsive expiration after the penetration of foreign bodies, or to dust from the feed (Bigoteau), to stings and bites of parasites (*Pentastomum*, *Æstrus*), to laceration of the vena pterygoidea, or of the vena maxillaris interna in fracture of the condyloid process of the maxilla. Epistaxis also frequently occurs in fracture of the base of the skull. Another cause may be congestion of the head or stasis, especially in chronic diseases of the heart and lungs, also compression of the jugular vein.

Rare causes of epistaxis are teleangiectasia or angioma; the erosion of individual vessels in ulceration of the nasal mucosa commonly leads to hemorrhage from the nose, particularly also in glanderous ulcers. Rare causes of epistaxis are: laceration of the arteria pharyngea (Blaise), of the dilated arteria carotis interna, of the arteria maxillaris interna (Walley, Cadéac) or of the arteria nasalis (Marafon).

Epistaxis finally occurs not infrequently in the course of general acute infectious diseases (anthrax, purpura hemorrhagica, scorbutus, smallpox); also in blood diseases (leukemia, anemia).

**Symptoms.** There is a discharge of blood from one or both nares, either in the form of drops (epistaxis) or in a continuous thin stream or in larger amounts (rhinorrhagia); the blood is usually bright red, not foamy, perhaps mixed with a few large air bubbles. If nasal catarrh exists simultaneously, the blood may appear in the form of fine streaks in a mucoid or purulent discharge. If there is a more severe loss of blood, the patient is frightened, the pulse is small and frequent, the mucosæ are very pale and a fatal issue due to loss of blood may occur under intensification of the symptoms within eight to twelve hours, or only after five to eight days. In the majority of cases, however, hemorrhage ceases spontaneously after a time. We generally need consider only such hemorrhages as dangerous which occur frequently in anemic animals or which are due to serious underlying infections.

**Diagnosis.** It is generally easy to recognize the nasal cavities or the accessory nasal cavities as the source of a hemorrhage. However, the cause of such hemorrhage can only be determined after a consideration of all concomitant circumstances and after a careful inspection of the nasal cavities, with the rhinolaryngoscope when possible. If there is a more severe hemorrhage such inspection must, however, be postponed, and the stopping of the flow of blood is the first requirement. In hemorrhage from the lungs the blood is likewise bright red; but it is finely foamy and there is cough and frequently dyspnea. Exclusion of hemorrhage from the lungs may be difficult in those cases in which portions of blood get into the pharynx and from there into the trachea or bronchi, causing cough and becoming more or less foamy. In hemorrhage from the stomach the blood is dirty brown, has an acid reaction and may in horses, and occasionally in cattle, be expelled from the nose with vomitory movements. Blood oozing out of ulcers is mixed with mucoid, purulent or ichorous discharges.

**Treatment.** Hemorrhage frequently stops soon and spontaneously if the animal is taken to a quiet place. If it persists, cold applications to the frontal region and to the nose are indicated, also astringent irrigations of the bleeding nasal cavity (alum, tannic acid, iron sulphate, chloride of iron in 3-5% solutions). In severe hemorrhage, which cannot be controlled otherwise, one or both nasal cavities must be tamponed; this may be done with absorbent cotton soaked in an 8-10% solution of chloride of iron, or with oil of turpentine or with a 0.01-0.02%

solution of adrenalin or suprarenin. The tampons so soaked must be wrapped in gauze and tied to a string, so that they can subsequently be removed safely. (If in horses or cattle both nares have to be closed by tampons, it is necessary to make a preliminary tracheotomy.) Sometimes ergotin given subcutaneously is very serviceable. In a case of Pease obstinate hemorrhage from the nose was controlled by the intravenous injection of a 2% solution of gelatin in a 0.4% solution of sodium chloride. Hemorrhages from varicose veins or from ulcers may be stopped by cauterization with silver nitrate or by the actual cautery. In very restless horses, narcosis may become necessary in order to secure the arrestment of hemorrhage. (For the treatment of a posthemorrhagic anemia, see Vol. I.)

**Thrombosis of the nasal veins** may occur in passive congestion, in marasmus, occasionally also in phlebitis (Zschokke). It is of some clinical importance, since occasionally it may be confounded with glanders. Corresponding to the thrombi one sees on the mucosa of the septum yellowish or yellowish gray nodules or narrow ridges, the former sometimes arranged like a rosary. The mucosa in their neighborhood appears dark red. The appearance of the nodules, the absence of ulcerations and the lack of swelling of the submaxillary lymph glands are sufficiently characteristic to exclude glanders.

Zschokke saw abnormally intense congestion in the veins of the nasal septum causing nasal stenosis in a horse in the course of thrombotic colic. The horse was breathing with difficulty through the nose and also through the opened mouth and made a strong snuffing noise. The nasal mucosa was intensely cyanotic, and foamy blood was oozing out of both nostrils.

## 2. Nasal Catarrh. *Catarrhus narium.*

(*Rhinitis catarrhalis, Coryza, Ozæna.*)

**Etiology.** One of the most frequent causes of acute nasal catarrh is a cold, especially in spring and fall, contracted during cold, damp weather, particularly during a sudden change from warm to cold. Other frequent causes are the inhalation of air which is irritating to the mucosa, dust of the street, dust of feed, moulds, sometimes the pollen of flowers (hay fever) or irritating vapors, viz., ammonia which has accumulated in poorly ventilated stables. (Stazzi discovered, in two cases in dogs, *aspergillus fumigatus* as the cause of nasal catarrh.) Similar are the effects of too hot air. In conflagrations the effect of the latter is, however, intensified by the inhaled smoke and soot.

Foreign bodies introduced into the nasal cavities intentionally or accidentally may cause catarrh or superficial or deep loss of substance. (Nunn saw an ear of wheat drop from the catarrhal nasal cavity of a steer; Parson removed a sponge from the nose of a horse.) The same is true of frequent injury of the nasal mucosa of the horse, which is often produced intentionally in order to mask existing glanders.



Infection appears to play a rôle in some cases. It does not appear unlikely that cold is a predisposing cause in lowering the resisting power of the nasal mucosa.

Nasal catarrh occurs secondarily in some general acute infectious diseases and in other inflammatory conditions of the respiratory organs; it also usually accompanies catarrh of the pharynx and larynx. Inflammatory processes of parts in the immediate neighborhood of the nasal cavities, especially in the bones, teeth, and alveoli of the teeth, usually extend into the nasal mucosa.

**Chronic nasal catarrh** develops from the acute form only very exceptionally; it is, as a rule, seen as a secondary affection to disease in the immediate neighborhood or in other portions of the respiratory tract. The most common affections leading secondarily to chronic nasal catarrh are: glanders, neoplasms, animal parasites (*Pentastomum*, *Cestrus*), catarrh of the accessory cavities, rachitis, chronic bronchial catarrh, pulmonary tuberculosis, lungworm disease, etc.

**Symptoms.** Acute nasal catarrh usually begins with local symptoms unless it be due to a general infection. One rarely sees a prodromal depression of the sensorium, malaise and moderate elevation of temperature. Smaller animals generally sneeze a good deal in the beginning, shake their heads and rub their noses against their feet or against the floor, while larger animals snort often. The mucosa appears intensely reddened and feels warm. Soon there is a discharge, usually from both nostrils. It is at first perfectly clear, thin fluid, watery; soon, however, it becomes tenacious, glassy, somewhat turbid, at the same time more abundant. Finally, in consequence of the admixture of numerous pus corpuscles, the secretion becomes purulent and less abundant; it dries at the margins of the nostrils and there forms crusts. Later on the discharge again becomes clear and then ceases. The mucosa, which is at first dry, gradually becomes shining. It retains its intense red color during the whole time and becomes more or less swollen; sometimes in the larger animals, usually in the smaller ones, respiration becomes snorting and sniffing. In dogs asthmatic attacks occasionally occur, due to a periodic accumulation of mucus between the turbinated bones (*Liénaux*). Sometimes rattling nasal sounds are heard. Exceptionally roundish, superficial erosions are formed on the mucosa. The intensely red base of these erosions soon becomes covered by new epithelium.

In rare cases there are formed thin-walled vesicles of millet to lentil-size (so-called "*Blatterdruse*," rhinitis phlyctenulosa). The process which leads to the formation of such vesicles is a rapid mucoid degeneration of epithelial cells or the accumulation of a serous exudate under the epithelial covering, which elevates it in spots from the underlying tissue. The vesicles later on dry and form crusts which are shed during the rapid regeneration of the epithelium.

One of the most common complications is acute conjunctivitis, manifesting itself by redness and swelling of the conjunctivæ, photophobia, and increased lacrimation. Some patients show difficulty in deglutition, also acute laryngitis, as a complication. The submaxillary lymph glands are sometimes slightly swollen and somewhat tender. The swelling soon disappears with the cessation of the nasal discharge.

The course of the disease is usually favorable. After the secretion has become purulent, the irritation of the mucosa ceases, the discharge rapidly diminishes, and recovery is complete within one or one and a half weeks.

**Chronic nasal catarrh** sometimes begins with the symptoms of acute nasal catarrh, which—with remissions—may exist for a long time. In other cases, the affection develops with chronic characteristics from the start.

The nasal discharge, which always exists, varies in its composition from case to case and even from time to time in the same animal. It is sometimes more mucoid, tenacious and glassy, sometimes more purulent; it may even be bloody or ichorous and fetid (ozena). It is usually not abundant, disappears from time to time entirely, to become again suddenly profuse, and is discharged especially after exercise, sometimes also during lowering of the head (rhinorrhœa, blenorrhœa nasalis). The secretion partly dries at the margins of the nares, it partly runs over the upper lip and leads to the formation of unpigmented streaks in consequence of maceration of the skin.

The mucosa appears bluish or brownish-red, or sometimes grayish-red, evenly swollen or uneven (rhinitis proliferata). Cicatrices of variable type may also be formed.

Catarrhal ulcerations as described above occur more frequently and in larger numbers than in acute catarrh. The swelling of the mucosa may cause stenosis of the nasal cavities and may bring about difficulty in respiration with snorting and sniffing. Narrowing of the nasal cavities occasionally is brought about by masses of exudate which accumulate between the turbinated bones (rhinitis concharum). In such cases thinning and deviation of the nasal septum may occur. The accumulation of masses of secretion and the asthmatic attacks caused by them are seen most frequently in dogs with short noses, and especially during or after exercise. The attacks manifest themselves by snoring inspirations (Hébrant and Hermans).

In horses we occasionally see nodular or smooth, rarely ulcerated, somewhat mottled, translucent elevations in the lower third of the nose, which occur in consequence of cellular proliferation and infiltration of all the layers of the mucosa as well as of the vessels and of the glands (hyperplasia mucosæ narium). These formations partially give the reaction of amyloid material. They do not extend into the neighboring



skin and, aside from their etiology, differ in this respect from rhinoscleroma, the occurrence of which among animals has not yet been established beyond a doubt, though some cases have been reported in literature.

In poorly nourished, anemic, weakly sheep, and especially in lambs, chronic nasal catarrh occurring after drenching and cold often assumes a severe type. In such cases the nose discharges a yellowish or dirty gray, sometimes fetid secretion, and respiration becomes snorting and rattling. Later on there generally follows an obstinate catarrh of the eyes and of the deeper respiratory tracts; the animals become greatly exhausted. It is impossible to decide whether this infection stands in any relation to the chronic form of hemorrhagic septicemia.

Cats sometimes develop a chronic nasal catarrh of unknown origin which leads to enlargement of the nasal bones, thickening of the skin of the nose and to swelling of the submaxillary glands.

Chronic nasal catarrh frequently extends to the neighboring accessory cavities, in horses, especially, to the antrum of Highmore or to the air sacs. In dogs generally, and especially in animals with short noses, catarrh often spreads into the frontal sinuses (Hébrant and Hermans).

The submaxillary lymph glands usually become somewhat swollen, tougher and harder, but they do not become adherent to the neighboring tissues.

**Diagnosis** is generally easy. The decision as to whether catarrh is primary or secondary is, however, more difficult. Since it may be of great importance to ascertain the exact cause of the affection, artificial illumination of the nasal cavities is indicated eventually by the aid of a rhinolaryngoscope or of the panelectroscope of Polansky-Schindelka, in certain cases also trepanation of the nasal cavity and a consideration of all concomitant conditions.

It is necessary to consider more particularly the catarrh of the antrum of Highmore and glanders. The former is indicated by unilateral, frequently fetid, discharge, tenderness to pressure of the infra-orbital region and sometimes by an abnormal prominence of the external wall of the cavity of the superior maxillary bone. In glanders are noted the peculiar nodules and ulcers and the condition of the glands at the entrance of the larynx. (See Vol. I.) If characteristic symptoms are absent the exclusion of the above indicated affections may be very difficult. However, trepanation, the mallein test, the agglutination or the complement-fixation tests will reveal the nature of the disease, even if clinical symptoms fail to do so. Increase of the nasal discharge, especially during work or in depression of the head, creates a suspicion of a catarrhal affection in a nasal accessory cavity or in the air sac. This may, however, also be observed in nasal catarrh with accumulation of the secretion between the turbinated bones. Acute nasal catarrh in horses sometimes represents the first stage of strangles; but the affection of the lymph glands will soon make the nature of the dis-

ease evident. Strangles may lead simply to the symptoms of acute nasal catarrh and the specific nature of the disease may only be recognized because other animals show the typical picture of the disease. (See Vol. I.) In other animals we must likewise consider the occurrence of specific nasal catarrhs.

**Treatment.** Acute nasal catarrh usually ends in recovery without any treatment. It is sufficient to protect the sick animals against drafts of air, to keep them in a moderately warm place and to give them feed free from dust. In severe cases with higher fever, with abundant discharge and snorting respirations, it is well to irrigate the nasal cavities with pure water, 1-2% solution of carbonate of soda or creolin, or make the animal inhale medicated vapors. When the secretion is very abundant, turpentine (1-5% evaporated over hot water) is very serviceable. Crusts adherent to the margins of the nares should be removed with warm water and the place should then be covered with fat or vaseline.

Chronic nasal catarrh requires local treatment with inhalation and irrigation of the nasal cavities.

In larger animals **inhalation** is brought about by placing under the nares of the patient a vessel filled with hot water and by wrapping a large cloth around the head of the animal and the vessel. If a horse should be frightened by this manipulation, the lower part of its head is placed into a feed sack, fastened to the head; the bottom of the feed sack is removed and the former connected with the vessel with hot water (Johns). Smaller animals may be made to inhale in a similar manner, or by the aid of a Siegel, a Bulling or a Wasmuth apparatus. The use of these apparatuses appears less serviceable however, since the greater portion of the sprayed fluid becomes deposited in the lower portions of the nasal cavities (Poeschel, Freund).

Much better results are obtained in all animals from **irrigation of the nasal cavities** with the aid of a syringe connected with a proper rubber tube, perforated in several places at its free end. This tube is introduced high up into the nares and the fluid is injected under moderate pressure. A catheter with lateral perforations connected with an elevated irrigator may also be used. A Frick or a Bayer-Kieselbach spray apparatus which may be introduced into the nares of a horse without difficulty is also quite serviceable. The following solutions may be used for nasal irrigations: 1 to 2% solution of carbonate of soda or creolin;  $\frac{1}{2}$  to 1% solution of carbolic acid, tannic acid; 2 to 4% boracic acid. Gorodtschaninow produced recovery in a short time in a case of chronic nasal catarrh by irrigation with alcoholic tannoform and menthol solution, composed of 2 parts of tannoform, 0.3 parts of menthol and 145 parts of 94% alcohol, plus 45 parts of distilled water. This solution was mixed before use with equal parts of distilled water.

However, in chronic catarrh of the uppermost portion of the nose these methods are insufficient, and here as well as in necrosis of the turbinated bones there is no other procedure left but trephining of the nasal cavity and irrigation from above with disinfectant and astringent fluids.

Internal medication appears superfluous; sulphate of atropine may perhaps be used in case of abundant secretion. In secondary nasal catarrh the primary affection must be subjected to the proper treatment, which is usually surgical. In empyema of the upper turbinated bones the removal of these parts sometimes becomes necessary.



**Nasal Catarrh with Cutaneous Desquamation in the Neighborhood of the Nose in Horses.** This disease, occurring frequently among horses, is probably due to noxious feed, in such a manner that certain irritating substances of such food, during ingestion, come in contact with the skin in the neighborhood of the nose, the mouth and the mucosa of the nose. Varying with the nature of the feed either the changes of the mucosa of the muzzle are more marked or, on the contrary, those of the nose. An origin as indicated above was established by Oekrész who found fifty-eight out of sixty-four horses of one owner affected seven days after they had been fed with chaff from a certain source. The disease did not spread to other horses. Aside from the changes of catarrh of the mouth and nose, desquamation of the skin was seen at the lower part of the head, the pigmented epidermis was shed in thin, but fairly large, lamellæ and flakes, and the skin assumed a peculiarly ragged appearance. Change of feed in such cases, supported by the usual therapeutic applications to the skin of borovaselein, brings about recovery within a few days (Oekrész, A. L., 1909, 514).

**Benign Infectious Nasal Catarrh in Cattle.** This disease, by Dieckerhoff also called ephemeral infectious nasal catarrh, is contagious in nature and is usually observed in adult cattle, only exceptionally in calves. It often occurs as a stable epidemic, frequently, however, also sporadically. According to Dieckerhoff, the period of incubation is, as a rule, only two days.

The relation of this disease to malignant catarrhal fever or croup of cattle (see Vol. I) and to infectious catarrh of the upper respiratory passages in cattle (see catarrh of the larynx and bronchi) is still to be established by further observations.

The first symptoms consist in decrease or even in complete lack of appetite, also weakness and a more or less febrile temperature, which, in severe cases, rises up to 40° C. and higher. In milch cows there is a decrease of milk secretion, lacerimation, swelling of the eyelids, a thin mucoid secretion from the nose, reddening of the conjunctivæ and of the nasal mucosa.

The course is acute and recovery occurs in adult cattle generally after one to one and a half days, more rarely after two to three days. Dieckerhoff observed a fatal case in a calf.

**Treatment** consists in a proper regulation of the diet (good hay and bran mash).

**Literature.** Dieckerhoff: *Specielle Pathologie*, 1892, ii, 86.

### 3. Contagious Nasal Catarrh of Birds. *Coryza avium contagiosa*.

(*Ansteckender Schnupfen der Voegel* [German]; *Coryza contagieux, morve ou roupie des poules* [French].)

Contagious nasal catarrh of fowls is an acute infectious disease characterized anatomically by a catarrhal inflammation of the mucous membranes of the head.

**Occurrence.** The disease occurs preferably among young fowls, especially chickens, during damp, cold weather, in fall

or spring. It is epizootic in character and often causes the death of many birds. Its economic importance depends both upon the great mortality and upon the fact that affected adult hens are unfavorably influenced as to their egg-laying capacity.

**Etiology.** Contagious nasal catarrh of fowls is due to an infection. This is shown not only by the epizootic occurrence but also by inoculation experiments (authors' observations), and by the observation that the disease is often imported through newly bought fowls (Ammenschlaeger, Feld). The nature of the infectious virus has not been determined exactly, but it is contained in the secretions of the mucosa of the head.

L. Gallez and later on Gratia & Liénaux found in the mucous secretion from the nose of sick chickens an organism similar to the diphtheria bacillus of Klebs-Loeffer. Gallez believed these bacilli to be an attenuated type of the diphtheria bacillus, while Gratia & Liénaux consider it doubtful whether the organism is an attenuated diphtheria bacillus or a pseudodiphtheria bacillus. Deich demonstrated spirilla-like organisms and also short bacilli rounded at both ends and occurring in groups.

The disease has formerly, but also again lately, been considered as a special clinical variety of fowl diphtheria (Colin, Schrevens, Gallez, Klee) due to the bacillus of fowl diphtheria.

**Natural infection** takes place by scattering the infected nasal secretion by movements of the head and in sneezing of the sick birds and by the ingestion of these secretions with contaminated food or water by healthy birds. Healthy flocks usually become infected by the importation of infected birds. The disease may, however, be spread just as in fowl diphtheria, by free flying birds coming from a distance.

Predisposing causes may likewise play a rôle. Damp and cold weather in the fall and spring, the sojourn in cold and drafty places, may assist in bringing about the infection.

**Susceptibility.** Young birds or such debilitated from any cause, especially young chickens, are affected most commonly. In some epidemics birds are affected without reference to age.

Deich did not succeed in the experimental transmission of the disease to chickens and ducks.

**Anatomical Changes.** Aside from emaciation and anemia a glassy or purulent mucus is found in the conjunctival sac, in the nasal cavities and in the pharynx. The mucosa of these parts is reddened. Pseudomembranous deposits or other organic changes are absent.

**Symptoms.** The disease begins with malaise. The sick animals separate themselves from others and sit quietly with



ruffled feathers and drooped wings. The appetite is decreased after one or two days, lacerimation occurs, also frequent sneezing and shaking of the head. At first watery, later on tenacious, whitish to yellowish masses are discharged from the nose. Such secretions are discharged from both nasal openings, and they dry there to yellowish crusts which close up the openings. The birds breathe through the open bill or respiration is rattling or sniffling. The eyes are kept closed and the lids become matted together by masses of secretion; in the conjunctival sacs a whitish, tenacious, however not croupous, exudate collects which makes the eyelids protrude. The cornea appears cloudy. On account of the matting together of the eyelids the sick birds are unable to take up their food and they may actually starve to death. The infra-orbital cell, which is filled with masses of exudate, sometimes protrudes in the shape of a swelling or tumor. In the pharynx we find a muco-serous exudate and intense reddening of the mucosa. Swallowing may become difficult. The birds frequently succumb with rapid emaciation.

Haubold saw in a goose-fattening establishment numerous cases of a disease resembling contagious nasal catarrh and leading to serious economic losses. After an incubation period of eight to sixteen days the birds showed reddening of the bill and a mucous nasal secretion which easily dried into crusts. The birds frequently scratched their bills, dipped them often into water, curved their necks occasionally or bent their heads toward the back, shook them, cried out from time to time, turned around in a circle and finally fell down almost unconscious, soon to get up again. Beginning with the third week there is diminution of appetite. Sometimes 10 to 15 per cent of the number of all birds succumb to the disease. It appears that it has been imported into Germany from Russia. Schreiber claims to have found as the cause of the disease a micrococcus, and its toxins are said to affect the cerebellum.

**Course and Prognosis.** The disease spreads rapidly among the birds of one establishment and leads to death in some epizootics after only one to three days; generally, however, the course lasts several days, up to three and six weeks. Most young birds die from the disease (sometimes there are 95 per cent of fatal cases); in adult birds the disease frequently ends in recovery.

**Diagnosis.** The clinical diagnosis is based on the contagious nature of the disease and the mucopurulent character of the catarrh of the mucous membranes of the head. Simple nasal catarrh, which under favorable hygienic conditions may also occur among a number of birds, may be distinguished by the fact that it does not spread widely, that the nasal secretion remains thin, fluid, and that the affection of the conjunctivæ and pharyngeal mucosa, which may coexist, remains moder-

ate in degree. In fowl diphtheria pseudomembranes are found on the mucosæ and also on the skin.

**Treatment.** Zürn recommends the inhalation of vapors of tar; Azary, irrigation of the nasal cavities with a 3-5 per cent solution of chlorate of potash, or a 5 per cent solution of borax; Klee advocates irrigation of the nasal cavities with a 2 per cent solution of sulphate of copper. The same solution may be used to brush the conjunctivæ and the mucosa of the pharynx. One should also see that the masses of secretion agglutinating the eyes and the nostrils are removed from time to time. The infra-orbital cell, if swollen, must early be laid open by an incision and must be irrigated for several days after the removal of the exudation. One must also attend to artificial feeding of young birds whose eyes have become closed and who consequently cannot feed properly. Feld recommends that white bread soaked in water or milk be introduced into the bill of such patients. While the disease lasts the animals should be kept in warm places.

**Prophylaxis.** Since treatment of the disease frequently is without results it is advisable to limit the spread of the disease by isolation of the sick, or better still, of the healthy birds, and by cleaning and disinfecting the coops and runs. The spread of the disease may sometimes be checked by the immediate slaughter of all the sick birds (Ammerschlaeger). The importation of the disease may be prevented by careful examination of newly bought birds and by keeping them in quarantine for some time.

**Literature.** Ammerschlaeger, W. F. Th., 1906, 27.—Deich, S. B., 1903, 67.—Feld, Leipzig, Geflüztg., 1906.—Gratia & Liénaux, Ann., 1898, 401.—Haubold, S. B., 1908, 76.—Klee, Geflügelkrkt., 1905, 18; P. VC, 1901, ii, 26.

#### 4. Infectious Nasal Catarrh of Swine. Rhinitis infectiosa suum.

(*Schnüffelkrankheit, Bösartiger Nasenkatarrh der Schweine* [ANACKER] [German]; *Rhinitis infectiosa* [IMMINGER].)

Infectious nasal catarrh of swine is an enzootic, infectious disease of young pigs, caused by the bacillus pyocyaneus, characterized anatomically by a hemorrhagic inflammation of the nasal and ethmoid mucosa and by hemorrhagic meningitis.

**Historical.** The disease has been known in Germany for a long time under the name of "snuffling disease"; it was believed to be a rachitic affection of the bones of the face or osteomalacia (see Vol. I) (Haubold, Harms, Wulff, Ostertag); others considered it a specific form of rhinitis (Schneider, Imminger); while still others (Anacker) thought that it was identical with malignant catarrhal fever of cattle or a form of scorbutus (Hering). Since nasal stenosis due to tuberculous



or actinomycotic changes has also been included under the term of "snuffling disease" Anacker, and particularly Friedberger & Fröhner, have advocated to abandon this name entirely. The authors are fully in accord with Friedberger & Fröhner and think that it is bad practice to throw together entirely different affections under one name merely on account of the presence of one common symptom; the name should therefore be abandoned entirely.

Imminger recognized the infectious nature of the disease in 1890 and Koske cleared up its etiology in all of its main points in 1906. Aside from these authors, Anacker, Schneider and others have furnished contributions to the symptomatology and pathologic anatomy of the disease.

**Occurrence.** Infectious rhinitis occurs during all seasons enzootically among pigs three to six months old; older hogs are affected only rarely. Wooden pig-pens appear to be particularly favorable to the appearance and to the spread of the disease; other observers claim that a rough stone floor favors the appearance of the affection. So far, only German veterinarians have reported on the appearance of this disease.

**Etiology.** *Bacillus pyocyaneus* is a usually small, slender, non-motile rod with rounded ends and with a single flagellum at the posterior pole; its length is between 0.6 to 6  $\mu$  and its shape is likewise very variable. It does not form spores. The bacilli have been found in the ethmoidal mucosa and in the brain of sick animals. They are easily stained with the watery anilin stains, but they are decolorized by Gram's method.

**Cultural Properties.** In a gelatin *stab* culture the bacilli develop almost exclusively in the upper part of the *stab* canal, to form first a depressed growth on the surface, greenish and fluorescent in color. Liquefaction spreads downward and the growth collects at the bottom of the liquefied gelatin as a slimy mass; the surface becomes covered with a greenish-yellow growth. On gelatin plates there are first formed small, roundish, yellowish-white colonies growing rapidly in size, their center darker yellowish and the periphery greenish fluorescent. On agar, there is formed a fairly thick, grayish-yellow covering, which at room temperature later on becomes deep dark green; the cultures emanate an aromatic odor resembling *jasmin*. On potatoes the growth is at first reddish-yellow and changes from the second day into green. Bouillon becomes after some time uniformly cloudy and greenish in the upper strata. Milk is coagulated.

**Tenacity.** The resistance of *bacillus pyocyaneus* is considerable, about equal to that of *staphylococcus pyogenes aureus* (Wassermann).

**Pathogenicity.** Small laboratory animals (mice, guinea-pigs) die after subcutaneous inoculation of pure cultures within two to five days; after intraperitoneal inoculation even after three to six hours. If young pigs are inoculated directly into the mucosa of the ethmoid, after a preliminary trepanation, they develop the same symptoms and anatomical changes as in



natural infection. According to the amount inoculated, the animals die after twenty-four hours or after eight to thirty days. Intramuscular inoculation leads to death in twenty-seven to thirty days. Intravenous injection of a bouillon culture only leads to a transitory elevation of temperature.

**Natural infection** occurs by the introduction of the bacilli through the nose. Since *bacillus pyocyaneus* is a common saprophyte, found especially in manure, straw, feces, etc., it easily gets into the noses of hogs in rooting. The inoculation experiments of Koske admit also the possibility of different portals of entrance. If the disease has once made its appearance it is spread by the nasal secretions of the sick animals.

Young animals are especially susceptible to natural infection.

**Pathogenesis.** After gaining access to the nasal mucosa, the bacilli multiply rapidly and soon get into the upper portions of the nasal cavities and into the ethmoid bone. They form powerful toxins and these produce, in the nasal mucosa and particularly in that of the ethmoid bone, a hemorrhagic inflammation which subsequently spreads to the meninges. Bacterial toxins, absorbed into the blood, soon cause a general elevation of temperature and petechiæ and ecchymoses in various portions of the body. Koske showed experimentally that the toxins of the *bacillus pyocyaneus* can by themselves produce a typical picture of the disease.

**Anatomical Changes.** The nasal mucosa is deep purplish red and the intensity of the redness is, according to Imminger, somewhat decreased upward. If the disease takes a short course one finds blood coagula in the ethmoidal cells; if the course has been more protracted a hemorrhagic purulent exudate is evident; there may be deviation and atrophy of the bony lamella. The frontal sinuses usually contain a serous fluid. Hemorrhages are seen under the periosteum of the ethmoid and vomer, as well as in the sheaths of the olfactory and optic nerves. The congested vessels of the meninges show blood extravasation; blood is also seen between the convolutions of the brain, sometimes in the shape of blood coagula. The substance of the brain appears edematous and the ventricles contain a red-tinged though clear fluid. Sometimes blood coagula are deposited on the choroid plexuses. While Imminger did not find any other changes, Koske found a turbid, reddish fluid in very small amounts in the peritoneal cavity, here and there diffuse reddening of the intestinal mucosa, streaky reddening of the cortical portion of the kidney, subserous hemorrhages and parenchymatous degeneration of the internal organs.

**Symptoms.** The disease is ushered in by a febrile elevation of temperature up to  $41^{\circ}$  and above and a diminution of appe-



tite; the sick animals feed very slowly or not at all, though they root a good deal in the food which is placed before them. They early show a certain degree of anxiety and excitement and make a peculiar blowing noise, as if they wanted to expel a foreign body which had gained entrance into the nose. They occasionally rub their snouts on their feed troughs or on the walls of the pens. Usually on the second day there is bleeding from the nose upon sneezing, and there appears a reddish, slimy, later on and in more protracted cases, a purulent nasal discharge; according to Anacker, even ichorous secretion flows from the nose in some cases. Breathing becomes more and more forced and snorting. If the nasal mucosa is much affected, there may be edematous swelling of the region of the nose and of the sub-maxillary glands. In the further course, generally after one to three days, marked cerebral symptoms become manifest, there is restlessness, which may increase to maniacal excitement and to attempts to scale the walls of the pens; also convulsions, which cause the animals to fall to the floor. The excitement is followed by coma and the animals lie apathetically in the straw, unable to rise.

**Course and Prognosis.** The disease generally takes an acute course and frequently ends fatally within from three to six days, under increase of the disturbances in respiration and of the general depression. In some epidemics all the animals die and it becomes necessary to slaughter the exposed animals in time before they show signs of the disease. Rarely do cases terminate fatally within one to two days. More frequently a chronic course is observed, the first tempestuous symptoms become milder, the appetite, however, remains poor or variable, there may be slight epistaxis at intervals and mild convulsions. The nutrition of the patients suffers gradually and the animals die from inanition after several weeks or months, unless they have previously been killed. Recovery is rare; different epidemics, however, vary greatly in this respect.

**Diagnosis.** The acute onset with high fever, the intense inflammation of the nasal mucosa and of the brain without protrusion of the bones of the face, furnish sufficient data for a diagnosis. Rachitic or osteomalatic extension of the facial bones is characterized by an afebrile course and by the marked disorder of the facial portion of the head with simultaneous protrusion of the hard palate toward the buccal cavity; there is in this affection a nasal catarrh only after a prolonged course and after a nasal stenosis has been existing for some time. Hog cholera, in its most acute form, might be confounded with contagious nasal catarrh because it is sometimes accompanied by epistaxis and by symptoms on the part of the brain. The absence of anatomical changes in the nose, and the changes found in the intestinal tract upon post-mortem examination, should prevent an error in diagnosis.

**Treatment.** This is frequently without success. It appears therefore best to slaughter most of the sick animals. If the symptoms are mild from the start, treatment should however be instituted. Imminger was successful in two cases with instillations of a 1% solution of corrosive sublimate, 1 tablespoonful into each nostril every one to two hours. One may also try irrigation of the nasal cavities with corrosive sublimate or another disinfectant solution. (See page 7.)

**Prophylaxis.** If the disease appears in a herd, it is well to separate the healthy animals and instill some corrosive sublimate or other disinfectant solution into their nares. The infected pens must be well disinfected and kept clean.

**Literature.** Anacker, *Spez. Path.*, 1879, 46.—Imminger, *W. f. Tk.*, 1890, 125.—Koske, *Arb. d. G. A.*, 1906, XXIII, 542.

### 5. Contagious Nasal Catarrh of Rabbits. Rhinitis contagiosa cuniculorum.

(*Influenzaartige Kaninchenseuche* [KRAUS, KASPAREK]; *Infectiöser oder Böartiger Schnupfen, Böartiges Schnupfenfieber, Böartiges Katarrhalfieber der Kaninchen, Kaninchenstaupe* [German]; *Rhinitis Purulenta* [ROGER & WEIL].)

Contagious rhinitis of rabbits consists in an enzootic contagious affection of the respiratory passages by a bacterium similar to the influenza bacillus.

**Historical.** This disease, which has long been known under the name of "malignant snuffles," was formerly believed to be a coccidia rhinitis of rabbits (see page 28). The investigations of Beck (1891), Kraus (1897), Roger & Weil (1901), Volk (1902) and Kasperek (1903), have, however, shown that the great majority of cases known under this or similar names is of bacterial origin. Affections similar clinically and anatomically have been observed by Suedmersen (1905) and Koppányi (1906), but they are due to bacteria of a different kind.

**Etiology.** The cause of the disease is a very small, slender, immotile bacillus of the size of the bacillus *bipolaris avi-septicus*, which is decolorized by Gram's method and which does not form spores.

**Cultivation.** In the presence of oxygen and at blood temperature the bacilli grow on all of the usual culture media. On gelatin plates there are formed after forty-eight hours small granular colonies with a sharp or, according to Kraus, serrated margin; the gelatin does not become liquefied. On the surface of agar there is developed a luxuriant grayish-white, iridescent growth. Milk is not coagulated, indol is not formed. The bacilli described by Beck, Kraus, Volk and Kasperek show some differences, mostly cultural in type, and according to Kasperek this points to the fact that different varieties exist.



The variations are, however, not considerable enough to justify a differentiation of the affections caused by the various types.

Suedmersen found a bacillus of the coli group in pleurisy of rabbits which is identical in its main characteristics with bacilli found by Kraus and Tartakowsky in a similar disease of guinea pigs.

The above-mentioned bacillus of Koppányi is distinguished by the fact that it is polymorphous, rather plump and surrounded by a capsule.

**Pathogenicity.** The bacillus is pathogenic for rabbits, guinea pigs, mice. Rabbits are most susceptible.

**Natural infection** usually occurs by inhalation of droplets of nasal secretion which are disseminated by sneezing, etc., by the sick animals and are inhaled by healthy rabbits, directly or later on with contaminated dust. The spread of the disease is also favored by feed contaminated with nasal secretion of sick animals, and by transmission through the hands of attendants. Rabbits are susceptible without reference to age.

**Pathogenesis.** After their entrance into the nose, the bacilli produce a violent inflammation of the mucosa of the nose and of the accessory cavities and they also get into the general blood circulation; there then occurs an elevation of the general temperature and in some epizootics, in fact, in most cases, an inflammation of the serous membranes and a pneumonia. The inflammation may spread from the nasal cavities into the deeper portions of the respiratory tract.

**Anatomical Changes.** The mucosa of the nose and pharynx appears intensely reddened, swollen and covered with purulent material. The accessory nasal cavities, frequently also the bronchi, contain a purulent exudate. In the pleura we find occasionally a serous, stringy or even purulent exudate, and the surface of the lungs is covered with a fibrinous deposit. The pericardium is rarely affected similarly. The lungs often show, aside from compression, atelectatic or bronchopneumonic foci. The specific bacilli are found in large numbers in the exudate of the mucosæ, in the other affected organs and in the blood.

**Symptoms.** Weakness and depression become apparent after a period of incubation of four to six days; the nares become moist and there is frequent sneezing. The temperature rises to 40° C. and above, and the appetite decreases. The nasal secretion is at first scanty and watery, or thick mucoid, and wets the hairs in the nasal region, the thorax and the anterior extremities; it becomes more abundant and purulent between the second and fifth days of the disease. Material of this kind is expelled by sneezing and snorting, and the animals rub their noses

with their front legs. The appetite diminishes more and more, there is emaciation and debility and also dyspnea and cough.

In pleurisy with pyemic cachexia of rabbits, as described by Koppányi, the clinical picture sometimes varies from the one described above. There occur very acute cases leading to death in two to three days, with rapidly increasing dyspnea and great prostration, high temperature, followed by a rapidly increasing subnormal temperature. Then there are frequently observed chronic cases with the formation of subcutaneous abscesses on various parts of the body or abscesses encapsulated in the internal cavities. After the spontaneous or artificial opening of such abscesses, the animals may recover, or more rarely they may die on account of acute exacerbations. Otitis interna sometimes occurs with oblique holding of the head or acute meningitis with symptoms of excitement (Barrat).

**Course and Prognosis.** In many cases the symptoms are intensified rapidly and the animals die within three to five days (acute type). In other cases, however, the symptoms are less acute from the start and the disease lasts fifteen to eighteen days (subacute form), or it lasts for a long time with mild symptoms (chronic form). All acute cases end fatally; a complete recovery is also seen rarely in the subacute or chronic variety.

**Diagnosis.** Characteristic for the disease are the severe local manifestations accompanied by high fever; Koppányi's disease of rabbits can usually be excluded only by a bacteriologic examination. In coccidia rhinitis, fever, prostration and dyspnea are less pronounced and microscopic examination shows the presence of coccidia in the nasal secretion; there is also no inflammation of the serous membranes.

**Treatment and Prophylaxis.** Since irrigation of the nasal cavity (see page 7) with 1% creolin or 3% boracic acid solution does not materially influence the course of the disease, our main efforts have to be made in the direction of prophylaxis. All sick animals ought to be killed at once and their cadavers must be destroyed. The cages and the barn, including the walls, should be thoroughly cleaned and disinfected; newly bought animals should be kept in quarantine and under observation for fourteen days.

**Literature.** Barret, *Rev. vét.*, 1908, 147.—Beck, *Z. f. Hyg.*, 1893, XV, 363.—Kasperek, O. M., 1892, 333.—Koppányi, *Z. f. Tm.*, 1907, XI, 429.—Kraus, *Z. f. Hyg.*, 1897, XXIV, 396.—Volk, *Centralb. f. Bakt.*, 1902, XXXI, 177.

## 6. Croupous Rhinitis. Rhinitis crouposa.

(*Nasal Croup.*)

Croupous rhinitis represents an intensely inflammatory process of the nasal mucosa, usually with the formation of extensive pseudomembranes.



**Occurrence.** Nasal croup is usually seen as an independent disease among horses, more rarely among cattle. The disease not infrequently appears enzootically among horses, particularly among the animals of a breeding establishment, and is then sometimes associated with follicular inflammation of the nasal mucosa (Roell).

**Etiology.** The disease sometimes appears after the inhalation of hot air and smoke in conflagrations. In most cases, however, an infection appears to be the causative factor. Roell observed the spread of the disease from sick to healthy horses. Obolenski, Berndt and Grunth observed a contagious spreading of nasal croup among cattle. (See Croup of Cattle, Vol. I.) The suspected infectious virus is not known as yet; in horses, however, the possibility that streptococcus equi is the etiologic factor cannot be entirely discarded. In one of the author's own cases this disease followed after streptococcus mastitis in a mare, and in a case of Wyssmann, in a cow, it was preceded by a parenchymatous mastitis.

Nasal croup is seen as a secondary affection in the course of some infectious disease (rinderpest, malignant catarrhal fever, morbus maculosus, strangles, etc.).

**Symptoms.** The disease is initiated with the symptoms of a severe acute nasal catarrh, followed soon by the formation of gray or reddish-gray pseudomembranes, several millimeters thick, which adhere more or less firmly to the intensely red and intensely swollen mucosa. These pseudomembranous deposits are either confined to smaller areas or they form larger, more extensive, continuous patches. They can be easily detached and after this has been done spots of mucosa are exposed to view which are void of epithelium, granular, intensely red and easily bleeding. After a few days the pseudomembranes become detached spontaneously. The epithelial covering is then replaced, the mucosa becomes gradually paler, and recovery takes place without leaving any permanent changes.

There is a yellowish, tenacious nasal secretion which is later mixed with shreds of pseudomembranes. Respiration is forced and snorting in severe cases. The soft parts of the nasal region, the submaxillary lymph glands and the afferent lymph vessels are more or less swollen, hot and tender. Very rarely we see in the neighborhood of the nares nodules and ulcers similar to those seen in follicular inflammation of the nasal mucosa. The body temperature is markedly elevated up to the shedding of the pseudomembranes.

The course is usually favorable; recovery occurs in about a week, provided that the other portions of the respiratory tract have not been affected.

**Treatment.** Treatment is similar to that employed in acute nasal catarrh. The shedding of the pseudomembranes may be

hastened by irrigation of the nose with a 1 or 2% solution of carbonate of sodium. Since the disease is often contagious, it is advisable to isolate the sick animals.

**Necrosis of the Nasal Mucosa.** It occurs very rarely as a primary disease after traumatic, chemical, thermal insults or after an infection (*bacillus necrophorus*); as a rule there are other underlying primary affections (*morbus maculosus*, acute glanders, also strangles, malignant catarrhal fever) which have first occurred. After the shedding of the necrotic portions of mucosa, deeply penetrating, dark red, or grayish-red, uneven ulcers are formed, with more or less elevated margins, around which the mucosa is inflamed. The submaxillary lymph glands are in a condition of acute swelling. The affection can usually be differentiated from glanders by the absence of glanders' nodules; sometimes, however, a differential diagnosis can only be made after a thorough bacteriological examination, as was shown in a case of Rabe.

## 7. Follicular Inflammation of the Nasal Mucosa.

### **Rhinitis follicularis.**

(*Rhinitis pustulosa*; *Coryza pustulosa equorum* [KITT].)

Follicular rhinitis is a peculiar inflammation of the nasal mucosa with nodule formation of the mucous glands, which nodules break down later on. The sebaceous glands of the skin of the region of the nose become similarly affected.

**Occurrence.** This is a disease of solipeds which occurs enzootically under conditions similar to those which are observed in croupous inflammation of the nasal mucosa.

**Etiology.** The disease undoubtedly owes its origin to an infection, as shown by the observations of its contagious nature by Roell and later by Friedberger & Fröhner. Kitt believes that *streptococcus equi* is the causative factor; Friedberger & Fröhner likewise believe that this is probable.

**Symptoms.** The disease begins with the symptoms of a violent nasal catarrh and as a rule with fever, whereupon, after two or three days, nodules, of the size of a millet seed, develop on the intensely reddened mucosa of the nasal septum. These nodules are quite numerous and they can be felt easily with the palpating finger. The nodules increase in size, become yellowish and frequently confluent and form a continuous yellowish surface; this disintegrates, becomes pale yellow and leaves after its removal small, roundish, shallow, intensely reddened ulcers with slightly infiltrated margins. The ulcers heal completely within a few days. In most cases similar nodules are developed on the skin of the alæ of the nose, the upper lips and the cheeks. These nodules also become yellow, ulcerate and



finally heal without leaving any traces. The lymph vessels, which drain the affected portions, are usually much swollen and can be felt as tender cords leading from the angles of the mouth backward, and also down toward the neck. In their neighborhood abscesses are formed here and there, which break open, but heal promptly. The submaxillary lymph glands are always in a condition of acute swelling. Sometimes one also sees nodules on the generally reddened conjunctiva of the eyes. The disease lasts two to four weeks and generally ends in complete recovery.

**Diagnosis.** The affection is distinguished from glanders in that the nodules and ulcers appear simultaneously or within a short time over a larger area in great numbers and that they heal without leaving any trace.

**Treatment.** Irrigation of the nasal cavities with mild disinfectant and astringent fluids (see page 7) and subsequent applications of desiccating ointments to affected cutaneous regions are very serviceable. Also applications of ointments containing mercury, iodoform, creolin, or iodine to the affected cutaneous regions.

## 8. New Growth in the Nasal Cavities. *Tumores narium.*

**Occurrence.** Neoplasms are generally rare in the nasal cavities. Most commonly are found so-called polyps, more rarely such other neoplasms as myxoma, sarcoma, carcinoma, angioma, osteoma, odontotomatoma, lipoma, ecchondroma; in the nasal cavities of cattle are also found mucons cysts. A similar clinical significance as true tumors have tuberculomata, as they occur rarely in the nasal cavities of cattle in the shape of nodular masses which are scattered over the surfaces, covered with a mucopurulent material and with yellowish spots. Gerspach saw in a horse tuberculous nodules of pea-size alternating with tuberculous ulcers covered with yellowish, dry adherent crusts. Very rarely actinomycomata are found in cattle in the shape of strawberry-like masses in the lower portions of the nasal cavities.

**Symptoms.** A tumor growing in the nose usually makes, in proportion to its size, one, rarely both nasal cavities more narrow, and thereby produces difficulty in respiration, blowing or snoring. According to the degree of stenosis, these symptoms are observed either during exercise or likewise during rest. If the stenosis is produced by a motile neoplasm, dyspnea and the noises accompanying it are observed only occasionally, or the character of the noises changes. If the nasal opening of the affected side is closed the noises cease, while closure of the healthy side increases them and increases dyspnea.

Differences in the volume of the air exhaled from either nostril may be ascertained by holding the moistened back of the hand in front of the nose or by placing a mirror in front of them and watching the deposit of dew, respectively, the difference in the size and the time of disappearance of the dewy deposit (Kaernbach). Labial breathing is observed in more severe cases. It disappears, however, on opening the mouth. With the exception of horses, animals breathe either temporarily or permanently through the open mouth and the respiratory noises mentioned above are then not heard. Tumors growing in the lower portions of the nasal cavities are detected by direct inspection, while those in the upper portions of the nasal cavities are felt upon sounding of the nasal passages.

There is usually a nasal secretion on account of the secondary nasal catarrh, which is, as a rule, unilateral, mucopurulent, often fetid, sometimes mixed with fragments of tissues. Soft or ulcerating neoplasms may lead to epistaxis, while tumors of the upper portion of the nasal cavities which extend into the pharynx may cause disturbances of deglutition. A unilateral, chronic swelling of the submaxillary lymph glands is rarely missed; however, it reaches a high degree only in the presence of malignant or tuberculous tumors. Sometimes a change of form of the noise is noted, or dullness on percussion of the ridge of the nose or exceptionally a protrusion of the hard palate toward the buccal cavity.

**Treatment.** Exceptionally only is any treatment, aside from surgical interference, serviceable. An angioma situated near the anterior nares may be made to shrink by brushing with a 10% solution of trichloroacetic acid.

**Literature.** Gerspach, Tuberculose eines Pferdes, Diss. Giessen, 1905.—Kaernbach, Die Neubildungen der Nasenhöhle und der Nebenhöhlen des Pferdes, Berlin, 1909 (Lit.).

## 9. Animal Parasites in the Nasal and Accessory Cavities.

### (a) *Æstrus ovis*.

(*Bremsenlarvenkrankheit*, *Æstruslarvenkrankheit*, *Hornwurmkrankheit*, *Bremsenschwindel*, *Schleuderkrankheit* [German]; *Vertige d'oestres*, *faux tournis* [French].)

The disease caused by the larva of the sheep fly is characterized anatomically by a catarrhal inflammation of the nasal and accessory cavities.

**Historical.** *Æstrus* larvae were first observed in the head cavities of sheep by Vallisneri in 1712; later on they frequently became the object of close studies; *Æstrus ovis* became well known from the descriptions of Bracy Clark (1797) and Hertwig (1838) and veterinarians have since then frequently discussed this subject.



**Occurrence.** The disease is found all over the world, excepting Australia, especially in young animals (yearlings); it occurs enzootically, more rarely sporadically. It is exceptionally seen in goats.

In fat-tailed sheep the larvæ of *Oestrus purpureus* likewise appear to occur. In the nose of buffaloes and camels the larvæ of *Oestrus maculatus* have been found, and in the pharynx of deer the larvæ of *Pharyngomyia picta* and *Cephalomyia rubibarbis*; in roes *O. stimulator*; in reindeer *O. trompe* and in moose *O. Ulrichii*.



Fig. 1. Larva of *Oestrus ovis* (seen from the posterior surface at the left; seen from the ventral surface at the right).

**Etiology.** The larvæ of *Oestrus* (*cephalomyia*) *ovis*, sheep fly, are, 2 to 30 mm. long, according to their varying stage of development. Their bodies are elongated and oval, flat on the ventral surface, convex on the dorsal and show ten or eleven rings. The youngest individuals are white, and transparent, the others yellowish white. The mature ones show a transverse striation of the rings; the anterior end is more elongated than the posterior one. The head ring carries two mouth hooks (Fig. 1).

The larvæ are developed from the ova of the sheep fly. They are 10 to 12 mm. long, yellowish-gray with transparent wings. The larvæ which leave the ova already in the oviduct are deposited by the female flies in the neighborhood of the anterior nares of the sheep, whence they wander into the nasal cavities, frontal sinuses, ethmoidal cells, and even higher up. They become adherent to the mucosa and progress in development. They mature after about ten months, i. e., in spring, migrate out of the middle nasal duct and change in the soil within twenty-four hours into pupæ. The fly leaves the latter after four to six weeks; after fertilization the females again deposit their ova in the neighborhood of the anterior nares of sheep.

Sheep flies swarm from the middle of May to October, for a shorter time in colder climates, especially during the noon hours. They rest preferably in the holes and clefts of sheep stables and among the brushes. From here the fertilized females hunt up herds of sheep to deposit the larvæ in the neighborhood of the anterior nares. On the approach of the flies; the sheep run together, put their heads together or place them on the ground, or hold their noses between their legs. If the larvæ are deposited in spite of this, and after they have begun to creep upward into the nasal cavities, the sheep become very restless, rub their heads on the soil or between their feet, shake themselves, sneeze frequently, and run around restlessly. The restlessness, however, soon disappears and the animals remain quiet for about ten months.

**Anatomical Changes.** The mature larvæ are, as a rule, found in large numbers (according to Zürn, 60 to 80) in the

frontal sinuses and in those at the base of the horns; sometimes, also in the antrum of Highmore and in the nasal cavities. They are strongly adherent to the mucosa which here shows small roundish depressions and in their neighborhood catarrhal changes. Exceptionally the brain and its membranes become invaded and likewise exceptionally the larvæ are found in the pharynx and larynx. They get there usually only after the death of the animal, but may exceptionally get there during life and cause death by suffocation.

**Symptoms.** Since the larvæ cause irritation of the mucosæ of the head only after maturity or after their migration, symptoms become manifest only in spring or in early summer. The disease usually begins with the mild symptoms of an acute nasal catarrh, which increases in intensity within the next weeks.

The first and most constant symptom is nasal secretion, frequently unilateral, at first clear, serous or seropurulent, later purulent; or possibly hemorrhagic. The animals frequently sneeze and blow and throw out an abundant secretion, occasionally also some larvæ. At the same time, the sheep rub their noses on their front legs, on neighboring objects and on the ground, so that the parts around the nose sometimes become denuded and subsequently edematous. The head is lowered from time to time, then suddenly elevated, bent energetically backward or laterally; the gait is staggering and uncertain. Simultaneous catarrh of the conjunctivæ leads to profuse lachrimation. The mature larvæ leave the nasal cavities after one to two weeks, and the nasal and conjunctival catarrh then disappear.

In a minority of cases the catarrhal symptoms are complicated by signs of excitement and depression. The gait of the sick animal becomes markedly disturbed; they raise their feet high, stagger with the hind legs and repeatedly fall down. There is depression of high degree and grating of the teeth, rolling of the eyes, and occasionally forced movements toward one side (Gilis) are observed. In such cases death usually occurs within five to eight days, exceptionally within three to four days after the first symptoms become manifest.

The intensity of the symptoms and the course of the disease depend largely upon the number of larvæ. They may be present in small numbers and they may, upon their migration, not produce any other symptoms except those of a catarrh. The location of the larvæ also has some influence upon the clinical picture. The invasion of the ethmoid bone, or the involvement of the meninges, or the migration of the larvæ into the cranial cavity, bring about serious nervous phenomena; nervous symptoms may sometimes develop on account of the affection of the frontal sinuses.

**Diagnosis.** The rare cases with forced movements may be confounded with giddiness of sheep. This, however, affects almost exclusively young animals, and forced movements



stand in the foreground of the clinical picture, while catarrhal signs are absent. The differential diagnosis, however, meets with great difficulties and becomes impossible when *Oestrus* larvæ situated high up lead to nervous symptoms, while symptoms of catarrhal affections of the mucosæ are absent, as does occur though in very rare cases. If larvæ of *Oestrus* are expelled with the nasal secretion, the diagnosis is always established. The disease can be differentiated from lungworm disease by the absence of cough, by the occasional presence of *Oestrus* larvæ in the nasal secretion, while lung worms and their ova are not found.

**Treatment.** The only rational procedure consists in opening up the affected cavities, removing the larvae that are present and accessible with a pair of forceps and anesthetizing those remaining with appropriate irrigations.

**Trephining of the Frontal Sinuses.** Since the larvæ are found preferably in the frontal sinuses, these must be laid open in particular. Zürn recommends as the place of trephining the two upper, Moussu, however, the lower angles formed by a line connecting the superciliary ridges with another line drawn at right angles into the middle of the former line. If the condition does not improve after trephining in this manner, it is advisable to remove the horns and thus to lay open the cavities at their base. After the larvæ have been recovered from the opened cavities, these must be irrigated with a fluid noxious to *Oestrus* larvæ. Serviceable fluids are: benzin diluted with water, oil of turpentine, 3% carbolic acid, 95% alcohol, lime water, etc. None of these fluids will kill the very resistant larvæ (Fischer), but they will cause them to contract strongly, so that they will leave their places of attachment and will be expelled from the nose by strong sneezing.

This treatment, however, can hardly be carried out when a large number of animals of a herd are affected and the operative procedure will then perhaps be employed only on the most valuable animals. The treatment sometimes does not have the desired effect and most animals, especially in the presence of severe cerebral symptoms, will, according to Zürn, remain sick and will succumb. Speedy killing of the animals is therefore indicated if the affection is violent, if trepanation cannot be carried out, or if, in spite of trepanation, the symptoms do not ameliorate.

The insufflation into the nose of tobacco, snuff, hellebore, all of which cause sneezing, the inhalation of irritating smoke, irrigation with various fluids (aside from those already named, vinegar or salt water), is frequently recommended. Considering the great resistance of the larvæ and their place of location, one cannot expect much success from such procedures.

**Prophylaxis** encounters very great difficulties. It is advisable to destroy the larvae. Sheep flies may be driven out of sheep stables temporarily by repeated fumigations. It also appears advisable not to pasture sheep on hot summer days in such locations where there are bushes and trees and to exterminate bushes from sheep pastures. Local conditions, however, usually prohibit the carrying out of such measures. It is therefore recommended to apply, in places where sheep

flies are common, ill smelling substances to the neighborhood of the nares of the sheep, such as tar, fetid animal oil, hartshorn oil, etc. This procedure, however, is likewise impracticable in the case of large herds. Under these conditions it is advisable, in such neighborhoods where the dangerous flies abound, to keep the sheep on hot summer days in fumigated barns and to allow them to visit the dangerous pastures only after sunset or at night.

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**Oestrus Disease in Dogs.** Ed. Sargent and E. Sargent (A. P., 1907, 392) have observed in Algeria, in such parts where the number of sheep is small compared with the number of inhabitants, that sheep flies deposit their ova into the eyes, lips and nose of shepherd dogs and of the shepherds themselves, particularly if they eat much sheep's or goat's cheese. The disease called "Thimmi" in the vernacular manifests itself soon after the deposit of the ova, after three to ten days, and during this time there is observed burning of the eyes, disturbance of vision, swelling of the conjunctivæ, lacrimation, also a serous nasal discharge, rubbing of the nose; occasionally symptoms of pain in deglutition, and cough depending upon affection of the pharynx and larynx. In the conjunctivæ and occasionally in vomited material one finds the very small white, lively, motile larvæ. Recovery occurs without exception. The larvæ do not attain full development. Tobacco smoke has proved effective in keeping sheep flies away from men and shepherd dogs.

#### (b) *Pentastomum Taenioides*.

**Historical.** This parasite was discovered in 1763 by Wrisberg; according to Zürn, however, before him by Chabert (1757); its life history was ascertained by Gurly, particularly, however, by Leuckart (1860).

**Occurrence.** The adult parasite is found preferably among the dogs of butchers, shepherds and hunters; it is found very rarely among other dogs, not at all among pet house dogs. The frequency of occurrence appears to be very variable in different parts of the world.

Among 630 dogs examined in Alfort with reference to this point Colin found the parasite among sixty-four (10.2%); in Toulouse only one (3.3%) out of thirty dogs examined showed the parasite; in Berlin (Deffke) thirteen (6.5%) of 200 dogs; in Chemnitz only 0.92% of 326 dogs examined (Tempel, Feureissen).

Aside from dogs the parasite has been found in other animals by Chabert, Schwammel, Leblanc, Rose in the nasal cavity of the horse; by Grève, in a mule; Rhind, in sheep; Bruckmueller, in the goat; Bremser, Colin and Miram, in the wolf; and by Laudon, in man. Kulugin found 1 to 2 mature individuals of the parasite in the nasal cavity of calves which five or six weeks previously had taken up ten to sixteen larvae with the food or drinking water.



**Etiology.** *Pentastomum taenioides*, Reed (*Linguatula taenioides*, Neumann) belongs to the *Linguatulidae* family of the *Arachnoidae* (Fig. 2); the body of the parasite is tongue- or lancet-shaped, wider at the anterior end, and gradually tapering at the posterior extremity. The external cuticle consists of about ninety rings and the margins of the body are serrated so that the parasite becomes similar to a tapeworm. The mouth, which opens at the anterior extremity, is surrounded by four hook-like stump feet. The male is 10 to 20 mm.; the female, however, is 8 to 10 cm. long. The ova (Fig. 3) are oval in shape, 90  $\mu$  long by 70  $\mu$  wide. Special organs of sense, of respiration and circulation are absent.



Fig. 2.  
*Pentastomum taenioides*.

The parasites grow from the larvae designated as *Pentastomum denticulatum* (*Linguatula denticulata*). These develop in the internal organs of herbivora. After the killing of the host they get into the nose of the dog, either directly or through the pharynx, or so that the ingested larvae perforate the wall of the stomach and migrate into the lungs, bronchi and finally into the nose (Gerlach). Herbivora infect themselves by the ingestion of feed which is accidentally contaminated by larvae. The latter develop further in the nasal or accessory cavities and mature in six to seven weeks. After copulation the female develops up to one-half million ova and these are discharged with the nasal secretion, contaminate grass or feed and are taken up by herbivora, in the body of which the larvae gradually develop. In the meantime the larvae occasionally appear to wander, getting into the lungs and bronchi, from which they are expelled with masses of secretion.

**Anatomical Changes.** The parasite is generally found in the upper portion of the median nasal ducts and in the cells of the ethmoid bone. It is found exceptionally in the frontal sinuses, in the lower portion of the nose and even in the pharynx (Gellé); the males are particularly liable to get into the larynx when wandering and hunting for the female. The number of parasites varies between one to eleven.

At the site where the parasites are adherent the nasal mucosa is usually reddened, swollen, covered with an abundant secretion or infiltrated with small hemorrhages. Only exceptionally do we find more intense inflammations; occasionally necrosis, especially in the cells of the ethmoid bone. (Concerning the changes produced in the internal organs by the larvae, see diseases of these organs.)

**Symptoms.** The *Pentastomum taenioides* sometimes does not produce any symptoms at all; it causes, however, in the

majority of cases, a chronic nasal catarrh accompanied by frequent sneezing (so-called *Pentastomum coryza*) and subsequent profuse discharge from the nose. The animals rub their noses on the ground or on their front legs or use their paws to rub over it. The nasal discharge is mucopurulent evidently mixed with blood and later on harbors numerous double contoured ova, each of which contains an embryo (Fig. 3). All symptoms are increased after running and sneezing; shaking of the head, rubbing of the nose may appear in the form of paroxysms, during which the animals may expel one or more parasites (Duméril, Mégnin). The olfactory sense is often intensely impaired. This is shown by the fact that the sick animals do not recognize their feed with the sense of smell and take it only if held before them.



Fig. 3. Ovum of *Pentastomum tacioides* according to Leuckart.

Sometimes there is restlessness and excitement, which is particularly noticeable during sleep, when the animals, which usually snore, sometimes jump up and sneeze convulsively. While awake restlessness is shown by the fact that the patients are irritable, growl, grind their teeth when approached, hide occasionally, also cry out, turn around in a circle, until they fall exhausted to the floor and lie there motionless. True maniacal attacks, as in rabies, hardly ever occur.

The difficulties in respiration may become so severe as to lead to attacks of suffocation; the facial expression betrays great anxiety, the eyes are staring, foamy fluid flows from the mouth, and respiration is accompanied by whistling and rattling sounds. Exceptionally a perforation of the hard palate may occur.

**Course.** The disease always takes a chronic course. The parasites may remain in the nasal ducts as long as fifteen months (Colin), but are finally expelled. Most dogs recover from the disease, but they frequently become emaciated, although finally they recover completely. Death is exceedingly rare and is then caused by the extension of an ethmoiditis into the meninges or by parasites getting into the larynx and causing death by suffocation (Dick).

In horses the exceedingly rare affection is characterized by frequent sneezing, shaking of the head, a mucoid or mucopurulent nasal discharge and chronic swelling of the submaxillary glands.

The **diagnosis** can be established beyond doubt only by the discovery of the parasites or their ova. Suspicion of *Pentastomum* invasion is however justified when symptoms of an obstinate chronic catarrh of the nasal and accessory cavities are observed, particularly in butchers', shepherds' or hunters'



dogs. The possibility of confounding the disease with rabies appears quite remote. The presence or absence of paralysis is of great differential diagnostic value; the presence of *Pentastomum* does, of course, not exclude the possibility of rabies.

**Treatment.** Therapeutic interference becomes necessary only in the presence of severe symptoms. Inhalations of chloroform, dilute ammonia or benzine and instillations of turpentine do not, as a rule, lead to satisfactory results. There is, therefore, nothing left when interference becomes indicated but to trephine the frontal sinuses or the nasal cavities in order to remove the parasites with forceps and to irrigate from above with a solution of creolin or carbolic acid.

**Literature.** Csokor Ö. Z. f. Vsk., N. F. I., 1887, 1.—Gerlach, H. Jb., 1869, 73.—Kitt, Bakterienkunde, 1908, 152.—Leuckart, Bau u. Entw. d. Pentast., 1860.—Neumann, Mal. paras., 1892, 542.—v. Rätz, Cbl. f. Bakt., 1893, XII, 329.—Zürn, Thier. Parasit., 1882, 105.

**Other Parasites in the Nose.** In southern countries horse leeches (*Haemopsis sanguisuga*) are sometimes found in the nose of the horse. In the nasal and infra-orbital cavities of birds the monostomum is very rarely found.—Ulreich (Vet., 1896, 292) found parasitic ova in the purulent nasal discharge of a young dog suffering from a bronchial catarrh, there probably identical with those of *Trichosoma aerophilum*, found by Creplin in the trachea and bronchi of a fox, by Müller in a badger and by Neumann in a cat. After intranasal and intratracheal injection of turpentine and oil of sesame, the disease ended in recovery in four weeks.

(c) **Nasal Catarrh in Rabbits Caused by Coccidia. Rhinitis coccidiosa cuniculorum.**

(*Psorospermia*, or *Gregarina Rhinitis* [ZÜRN].)

After the invasion of the nose of a rabbit by coccidia, there occurs an intense inflammation. The affection occurs enzootically and usually spreads to the other mucosae of the head.

**Historical.** All enzootic nasal catarrhs in rabbits were formerly known under the collective name of malignant coryza ("malignant snuffles"), but they were all classified as coccidial rhinitis after Zürn had demonstrated the presence of coccidia. Further observations, however, have shown that not all of those affections properly belong to coccidial rhinitis (see page 15).

**Etiology.** The invasion by coccidia (according to Railliet, *C. perforans*; however, according to Martin, *Eimeria s.*

*Coccidium cuniculi*) probably occurs in the same manner as does the infection by pathogenic bacteria which cause contagious rhinitis (see page 15). Dampness of the bins and the straw appears to be favorable to the appearance and spread of the disease. Young animals preferably are susceptible to the disease; older animals are usually affected slightly and temporarily.

**Symptoms.** The clinical picture of the disease is very similar to that of contagious rhinitis (see page 16). There is at first moisture around the nares and a scanty mucous secretion. At the same time there is sneezing and sniffing and the animals sometimes rub their noses with their front feet. There is slight fever and accelerated respiration. The sick animals are at first fairly lively and their appetite is good; but after a few days they become listless; they frequently grind their teeth and there is complete lack of appetite. In the meantime, the nasal discharge has become more profuse and the respiration becomes more and more difficult. Frequently, symptoms of conjunctivitis and stomatitis set in.

In those cases where the coccidia have invaded the middle ear from the pharynx, one sees oblique position of the head, staggering and rolling in consequence of the involvement of the semicircular canals; these disturbances may at first be only transitory, but become permanent later on. More intense external irritation may bring about convulsions. Later on there is profuse diarrhea, rapid emaciation, and finally death follows.

**Diagnosis.** Coccidial rhinitis can be diagnosed after the microscopic detection of coccidia, which are always found in enormous numbers in the nasal secretions or in the conjunctivæ. In this manner the affection can also be differentiated from bacterial contagious rhinitis. In mange of the ears catarrhal symptoms of the mucosæ of the head are absent.

**Treatment.** Local pathologic conditions may be treated by irrigation or applications of a 3 per cent boracic acid or a  $\frac{1}{2}$  to 1 per cent solution of sulphate of zinc or copper sulphate, or of a 1 per cent solution of creolin. Braun recommends in addition the internal administration of flores sulphuris with finely powdered sulphate of sodium in knife-point doses.

The prophylaxis is based upon the same principles as in bacterial contagious rhinitis (see page 17). Young animals should always be separated from the older ones as early as possible.

**Literature.** Braun, *Kaninchenkrkht.*, 1907, 50.—Martin, *Rev. vét.*, 1909, 278.—Bailliet, *Zool. méd.*, 1895, 141.—Zürn, *D. Z. f. Tm.*, 1878, I, 24.—Vortr. f. Tzts, 1878, I, 2 FL



**10. Catarrh of the Maxillary Sinus. Catarrhus antri Highmori.**

Inflammation of the mucosa of the antrum of Highmore, mostly chronic and catarrhal in character, usually leads to the accumulation of a mucopurulent exudate in the cavity of the superior maxillary bone. (Empyema sinus maxillaris sive antri Highmori.)

**Occurrence.** Catarrh of the antrum of Highmore is observed comparatively frequently in horses, rarely among other animals. Horses are generally affected when advanced in years.

Among the Prussian army horses during 1899-1908, 0.2% of the total number suffered from this affection, and compared with diseases of the respiratory tract, affections of the antrum were equal to 2% of the former.

**Etiology.** The disease rarely occurs as a primary affection, as after violent traumatic insults to the maxillary sinus. It is generally secondary to an extending nasal catarrh (see page 3), which spreads to the mucosa of the maxillary sinus which is continuous with that of the middle nasal space through a slit-like opening. Diseases of the teeth and alveoli are likewise frequently the primary cause of the affection, yet often the teeth themselves do not show any affection when inspected through the buccal cavity (Imminger). Caries of the teeth and alveolar inflammations, which are frequently found in horses and occasionally in cattle and dogs, often lead to a spread of the inflammatory process into the mucosa of the maxillary sinus or to an invasion by particles of feed.

In some infectious diseases there may also occur a catarrh of the antrum, as for instance in glanders, malignant catarrhal fever, infectious rhinitis of hogs (see page 11) and of rabbits (see page 15), in coccidia rhinitis (see page 28). Neoplasms (polyps, sarcomata, carcinomata, cysts) may also be causative factors, exceptionally botryomycosis or echinococcus.

**Anatomical Changes.** In the beginning of the affection there is noticeable only a more or less uniform or mottled redness and swelling of the mucosa, which later on becomes uneven, nodular, as if studded with millet seeds, and which also exhibits larger gelatinous nodules on sausage-like ridges. If the disease has lasted for a long time, wart-like formations appear on the surface of the indurated mucosa (F. Müller). The cavity of the maxilla contains a discharge which is usually purulent, occasionally fetid, and which may be mixed with particles of food; more rarely the exudate is serous (empyema, hydrops antri highmori sive sinus maxillaris); the walls may be covered with fungi (Nielsen found the mycelium of *mucor spinosus*). If the opening has become closed the cavity is

found completely filled with secretory masses. The anterior wall is thin and protruding. The wall separating the large from the small sinus in the horse may disappear completely. If the catarrh depends on a primary disease of the teeth, one generally finds, at the inner wall of the cavity, denuded rough bone, or one can feel the passive motility of a tooth through masses of granulation tissue.

**Symptoms.** In the horse the disease usually begins with the mild symptoms of a chronic unilateral nasal catarrh. There is at first a mucoid, then a mucopurulent, finally a purulent unilateral nasal secretion, which, in the course of time, macerates the upper lip (white streak!). The amount of secretion varies a good deal; it is usually increased after work and larger amounts may be voided on depressing the head, but the discharge may also cease entirely for a time. The nasal mucosa appears somewhat reddened.

An increased tenderness of the bony wall of the sinus can usually be demonstrated. In severe cases increased sensitiveness of the infra-orbital nerves may be present.

Dullness on percussion can be shown only rarely, i. e., when the catarrhal secretion or a tumor fills out a portion of the sinus situated above the facial ridge, or if a tumor is connected with the external wall, or if the mucosa is considerably thickened. In case of a partial thinning out of the bony wall by malignant tumors, the percussion sound may on the contrary be louder and deeper, because the thinner bone plate produces more extensive vibrations.

The percussion sound of the maxillary sinus is solely produced by the vibrations of the fairly elastic bone plate and is therefore duller only in those exceptional cases in which the vibrations are interfered with either by a high level of fluid or by a considerable increase in the thickness of the wall.

Changes in shape are quite rare. They can at first only be seen by a close comparison of the two sides of the face. Later on the deformities attain such a high degree that the disfiguration can already be observed at a distance. One can then usually also demonstrate that the bone is thinner and that it can be pressed in. Fluctuation can be shown in very advanced cases, and in these the respiration may be difficult and blowing.

The submaxillary lymph glands of the affected side become swollen in the course of time and are changed to tough hard nodules, which are not very painful or not sensitive at all and not adherent either to the skin or to the bone.

In **cattle** one usually observes unilateral mucoid or mucopurulent, sometimes bloody, nasal discharges, occasionally mixed with masses of fibrin. The infra-orbital region is tender to pressure. Here there may occasionally be dullness and



perhaps a protrusion of the wall. A fairly characteristic symptom is frequent blowing with violent movements of the head and the expulsion of mucopurulent masses from the nares. The respiration is snoring and accelerated, but it becomes normal again after the removal of the masses of secretion from the nasal cavities, which have accumulated there from the sinus.

**Dogs** frequently shake their heads and rub their noses with their front feet. There is also a unilateral seropurulent or mucopurulent nasal discharge, which may be mixed with blood and may be fetid. In the further course the respiration is blowing, due to constriction of the nasal canal. This disappears after opening the buccal cavity or after closing the affected side of the nose. Simultaneously with the appearance of respiratory difficulties, or before these occur, a swelling of the infra-orbital region becomes noticeable; it is tender to pressure, hot and later on fluctuating. If fluctuation has occurred, spontaneous rupture takes place with a diminution of the pressure symptoms which may have existed. The opening in the skin closes within a short time, but a new rupture occurs within one to two weeks and a fistula then persists (Hobday).

**Course.** With the exception of those cases of catarrh of the antrum of Highmore which are a part of the clinical manifestations of contagious rhinitis (see pages 11, 15), the affection usually comes on very slowly and it often takes months before it is fully developed. Even after the development of a grave condition the patients remain alive for months and are able to work. Later on, however, perhaps only after years, severe disturbances develop, especially respiratory difficulty, a kind of dumb-staggers, and symptoms of meningitis if the process has invaded the frontal and ethmoidal sinuses. In horses, the inflammation frequently spreads to the mucosa of the lacrimal duct (Plósz), and lacrimation and possibly conjunctivitis occur. The exudate may also break through an alveolar space and so lead to the formation of a fistulous tract. Bone necrosis may finally be observed. Especially in horses cases are often mild and may recover spontaneously (F. Müller, authors' observation).

The **diagnosis** is based upon the presence of a unilateral nasal secretion, unilateral swelling of the glands and the generally existing tenderness of the outer wall of the sinus. A diagnosis can generally be made even if a deformity is not yet present. For the exclusion of glanders, trephining, the mallein test or other immuno-diagnostic tests may be necessary. After opening the sinus, it is possible to ascertain whether a simple catarrh is present or whether it is secondary to another primary affection. Very fetid pus creates a suspicion of caries. If the latter is present one can generally detect

rough bone on the internal wall of the sinus or a corresponding passively movable molar. Tumors are palpable and frequently protrude through the wound. One of the authors' case, however proved that a diagnosis by the aid of trephining of a non-glanderous catarrh of the sinus could not remove the suspicion of simultaneously existing nasal glanders. Mucoïd degeneration of the bones of the face is distinguished from catarrh of the sinus by a relatively rapid occurrence of respiratory difficulties, marked deformity and softening of the superior maxilla; rachitis and osteomalacia by the fact that the changes are seen on both sides of the face and are not confined to the region of the antrum of Highmore.

**Treatment.** If catarrh of the antrum of Highmore has developed in connection with rhinitis, expectative procedures appear indicated for one to two weeks, since such cases frequently recover spontaneously. Otherwise trephining of the affected cavity followed by evacuation of the accumulated secretion and local treatment of the inflamed mucosa by energetic and repeated irrigation leads to recovery. Bad teeth must be removed. A number of disinfectant and astringent solutions may be used for irrigating the cavity; for instance, a 2 to 3 per cent solution of creolin or carbolic acid; 1:1000 corrosive sublimate, 1 per cent solution of alum, tannic acid and sulphate of iron; 0.1 per cent solution of silver nitrate. The irrigations have to be continued until the opening of the antrum is free and until no more pathologic secretion is produced. Appropriate treatment as indicated in simple catarrh always leads to complete recovery.

**Literature.** Hobday, V. J., 1905, 137.—Imminger, Verh. deutsch. Naturf., u. Aerzte, 1906.—Lange, B. & W., 1906, 193.—Mörkeberg, Maanedsskr., 1907, XIX, 92.—Müller, Monh., 1907, XVIII, 481.—Ries, Rec., 1899, 350.—Williams, Amer. v. R., 1906, 185.

**Myxoid Degeneration of the Turbinated Bones and of the Facial Bones in the Horse.** Foals sometimes develop a peculiar morbid process which leads to a myxoid degeneration of the turbinated and neighboring bones. The affection was first observed by Sand; similar cases have since been described by Deupser, Hützen, Nielsen, Fröhner and Johne.

The cause of the pathologic process is unknown. The disease has been observed only in foals and is characterized by a comparatively rapid development.

The first symptom is a nasal discharge which is mucopurulent or purulent, and which becomes more intense after a variable period of time. Simultaneously or somewhat later, especially during exercise, there is difficulty in respiration accompanied by a snoring sound, and these symptoms increase in intensity and become continuous. One observes also early a unilateral protrusion of the facial bones, especially of the superior maxilla; the swelling progresses rapidly and involves either the frontal or the masseter region. The swelling which is at

first of bony hardness later on becomes soft in the region of the greatest prominence, and subsequently in other places; finally there is distinct fluctuation. The laryngeal glands are always enlarged but not painful. Left to itself the affection generally leads to suffocation of the emaciated animals.

The disease does not always develop so rapidly, but after a few weeks it may be observed in an advanced stage. Sand saw the affection in a six-year-old gelding which had already snored some when a foal, but which manifested difficult respiration and swelling of the region of the antrum of Highmore only in its sixth year of life.

When the prominent region is incised or sawed into, a considerable amount of slimy serous fluid is discharged, mixed with yellow fibrin-like coagula. The antrum is considerably enlarged, the partition walls have disappeared and have been changed into a friable tissue. The internal surface of the cavity is covered with a soft gelatinous material which here and there contains sacs filled with mucus. The turbinated bones may likewise be enlarged and the nasal passages may be closed completely.

Microscopic examination shows that the major part of the bone has been changed into myxoid tissue and that lacunar absorption has taken place. In the neighborhood one sees an active new formation of bone.

The treatment consists in timely trepanation followed by disinfectant irrigations. This procedure usually leads to permanent recovery.

**Literature.** Fröhner, Monh., 1897, VIII, 514.—Sand, *ibid.*, 1893, IV, 193.

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**Inflammation of the Cella Infraorbitalis in Fowls.** This affection generally occurs in the course of diphtheria and of contagious nasal catarrh, exceptionally in consequence of the presence of parasites (monostoma); it leads to the formation of a hot, tender swelling in the infraorbital region (so-called swollen face of chickens). If pressure is made upon this swelling a mucopurulent exudate is discharged from the nasal opening of the same side. Splitting of the swelling in the long axis of the head, followed by irrigation, usually leads to recovery unless the primary affection takes an unfavorable course.

## 11. Catarrh of the Frontal Sinus. *Catarrhus sinus frontalis.*

Under the name of catarrh of the frontal sinus are designated more or less deeply penetrating, generally chronic, inflammatory processes in the mucosa of the frontal sinuses which lead to the formation of mucopurulent masses of exudate (empyema sinus frontalis).

**Occurrence.** Catarrh of the frontal sinus occurs only rarely in horses and dogs, more frequently in cattle. Railliet saw one case in a sheep.

**Etiology.** Traumatic insults causing a hemorrhage into the frontal sinus or a rupture or a fracture of the bony wall

of the frontal sinus or a fracture of the horn in cattle, usually play an etiologic rôle. An improper frontal yoke may be the cause of the disease. Joest described a case in a dog in which a plug of cotton was left in the frontal sinus after trepanation, and caused a purulent catarrh. In a certain number of cases catarrh of the frontal sinus developed secondarily as a sequel to catarrh of the maxillary antrum (in horses and possibly also in dogs), or as a sequel to malignant catarrhal fever and finally in consequence of neoplasms in the frontal cavities.

**Symptoms.** Corresponding with the usually unilateral affection we often see a unilateral, sometimes fetid nasal discharge, particularly after blowing or after an attack of cough brought on artificially, or after the animal has lowered its head. The discharge is often profuse and is partly licked up by the animals. The first symptom in cattle may be epistaxis, which is later on followed by other symptoms. Firm pressure upon the frontal bone, or percussion of the frontal region or of the base of the horns causes pain; occasionally one may elicit dullness on percussion on either one or both sides. Cattle hold the head obliquely in unilateral affection and lower it in bilateral disease. There may be unilateral or bilateral conjunctivitis with edema of the upper eyelids. In severe cases, particularly in cattle, there may be unconsciousness due either to dull pain in the frontal region (Moussu) or to pressure upon the brain in consequence of bulging of the internal bone plate into the cranial cavity. In the latter case, exceptionally also under other conditions, one may see the clinical picture of meningitis or of epileptiform attacks. Railliet saw in a sheep the symptoms of gid.

**Diagnosis.** The disease is easily diagnosed on the basis of the symptoms described, the most important of which are tenderness to pressure of the frontal region and of the base of the horns, elevation of temperature in these regions and nasal discharge.

**Treatment.** Trepanation of the frontal sinuses is the only proper treatment. Imminger, however, brought about recovery in horses by energetic irrigation by way of the trephined antrum of Highmore.

Moussu proposes that in cattle under three years of age, where the cavities at the base of the horns are not yet well developed and where the lower portion of the frontal sinuses do not much bulge, trepanation should be made in the upper portion of the frontal sinuses in the middle between the median line and the horn pegs. In older animals the core of the horn cavity should be opened 1 to 2 cm. outside of the core of the horn ring and also the lower portion of the frontal sinus, directly over the superciliary ridge, in the middle of the affected half of the front. Irrigation should be performed with lukewarm disinfectant and astringent solutions.

Trepanation in dogs has been described in detail by Weis & Parascandolo.

**Literature.** Imminger, W. f. Tk., 1908, 1.—Weis & Parascandolo, D. t. W., 1903, 17.—Mörkeberg, Maanedsskr., 1907, XIX, 92.—Railliet, Rec., 1881, 398.—(Compare also literature on catarrh of the antrum of Highmore.)



## 12. Catarrh of the Guttural Pouch. *Catarrhus sacci aerophori*.

Catarrh of the guttural pouch is a collective name for acute, or more commonly chronic inflammations of the mucosa of the guttural pouch, which lead to an accumulation of masses of secretion (*empyema sacci aerophori*).

**Etiology.** Catarrh of the guttural pouch is due only very exceptionally to traumatic insults (penetration of a ball or a splinter of the fractured lingual bone) or to the penetration of foreign bodies or particles of feed from the pharynx. Whether the moulds found by some authors in the guttural pouch were the primary cause of the disease is still an open question. Catarrh of the guttural pouch is as a rule a secondary affection due to catarrhal or more deeply penetrating inflammatory processes of the deeper portions of the nose or of the pharynx, the process spreading along the Eustachian tube. Exceptionally severe inflammation of the neighboring soft parts (parotid gland, retropharyngeal glands) may stand in an etiologic relation to catarrh of the guttural pouch. The latter affection is only rarely a part of the clinical picture of glanders.

**Anatomical Changes.** The mucosa is sometimes reddened, also covered with ulcers; if the case has been long standing, it is thickened and uneven; frequently mould colonies are found to have developed. The guttural pouch contains tenacious muco-purulent, occasionally putrid masses in varying amounts, which after desiccation are caseous or more firm (*concrementi sacci aerophori*) and which have wrongly been called chondroids or gutturaloliths. The cavity either contains one large mass of this type or a number of them (up to 200). Owing to decomposition of the secretion there may be a collection of gas (*meteorisms sacci aerophori*), exceptionally the guttural pouch may be filled with a serous fluid (*hydrops sacci aerophori*).

**Symptoms.** The clinical picture of catarrh of the guttural pouch, particularly in the early stage, is in part similar to that of catarrh of the antrum of Highmore. There is a unilateral mucopurulent or purely purulent nasal discharge, and a gradually increasing, not painful, swelling of the submaxillary lymph glands on the affected side. As the masses of secretion increase, however, the affected guttural pouch becomes enlarged in the course of time, and a protrusion in the parotid region becomes visible. The swelling is usually unilateral and may reach a variable size; it completely obliterates the depression back of the inferior maxilla, indeed a protrusion may be seen here (see Fig. 4). The latter is usually soft and doughy in consistency and can be made smaller by pressing and

kneading, while the discharge from the nose increases. The percussion sound is dull either over the whole of the parotid region or only over its inferior portion. If much gas has been formed the parotid region has an elastic, tense consistency and the percussion sound may be partly tympanitic. The rhinolaryngoscope shows in severe cases a protrusion towards the buccal cavity, variable in size, situated laterally to or in front of the arythenoid cartilages.

In a horse with catarrh of the guttural pouch and perforation of the membrane tympani of the ear of the same side Durante noticed a mucopurulent discharge from this ear which could be brought about at will by pressure on the diseased air sac.

The enlarged guttural pouch pushes aside and compresses the neighboring organs. It may particularly press the arythenoid cartilage upward or inward and in this manner cause laryngeal whistling. Pressure may increase to such an extent and may bring about such a degree of stenosis of the larynx that attacks of suffocation may come on even at rest, especially if the amount of air in the guttural pouch becomes temporarily increased. The swelling also presses the wall of the pharynx inward and in this manner produces difficulties in deglutition.



Fig. 4. Catarrh and meteorism of the guttural pouch of the horse. Intense swelling of the parotid region.

Zschokke saw in one case paralysis of the pharynx due to a spreading of the inflammatory process along the neighboring nerve trunks. In case of ulceration, unilateral epistaxis, and after erosion of neighboring larger vessels fatal hemorrhage may occur. Difficulties in deglutition may cause aspiration pneumonia and pulmonary gangrene. Such complications as will often occur after a longer duration of the affection make it one of serious importance.

**Treatment.** Recent cases can be cured by opening of the guttural pouch followed by repeated irrigation. If, however,

the enlargement of the guttural pouch is already quite considerable, and if the sac has become adherent to neighboring structures, the prognosis as to recovery is very bad. Irrigation of the guttural pouch with a Guenther catheter is not advisable on account of the danger of aspiration.

**Literature.** Cadiot, Bull., 1895, 219.—Johne & Uhlrich, S. B., 1831, 56.—Montané, Rev. vét., 1897, 397.—Schlampp, W. f. Tk., 1884, 21.—Savarese, Clin. vet., 1889, 309.—Thomassen, Ann., 1891, 121.

**Bloating of the Guttural Pouch.** (Tympanitis s. meteorismus sacci aerophori.) Bloating of the guttural pouch (meteorismus idiopathicus) is sometimes seen in foals as a primary affection. The cause of it is probably paralysis of the muscles of the Eustachian tube, in consequence of which active dilatation ceases, which causes the expulsion of air (Gerlach, Dieckerhoff, Peter). Exceptionally bloating of the guttural pouch may be due to congenital anomalies (the presence of folds of mucosa which close like valves [Niebuhr, Thomassen]). The affection occurs secondarily in consequence of chronic catarrh of the guttural pouch (meteorismus symptomaticus).

The **symptoms** are a puffy, elastic swelling in the region of the parotid (Fig. 4) which may be unilateral or bilateral; on percussion a tympanitic sound is elicited; the swelling may decrease upon pressure and a peculiar noise may then become audible. Cases are also observed in which the swelling is only found in the pharynx. Difficulty in respiration is always observed, difficulty in deglutition occasionally. In very serious cases pressure upon the bloated guttural pouch produces whistling or roaring sound during respiration. Nasal discharge or swelling of lymphatics are absent in idiopathic bloating of the guttural pouch.

The **treatment** consists in opening the guttural pouch, if necessary also in artificial dilatation of the tubal opening, and irrigation.

**Literature.** Cadéac, Journ. vét., 1908, 680.—Degive, Ann., 1900, 20.—Penberthy, J. of comp. Path., 1894, 174.—Peter, B. t. W., 1903, 618.



## SECTION II.

### DISEASES OF THE LARYNX.

#### 1. Catarrh of the Larynx. *Catarrhus laryngis.*

(*Laryngitis catarrhalis, Angina.*)

Catarrh of the larynx develops, at least in its acute form, as a superficial inflammation of the laryngeal mucosa in connection with catarrh of the trachea (tracheal-laryngeal catarrh); also in connection with catarrh of the pharynx (catarrh of the upper respiratory passages). These affections may be considered under the head of laryngitis because the symptoms of the latter stand in the foreground of the clinical picture.

**Etiology.** One of the most common causes of acute laryngeal catarrh is taking cold, hence the disease is frequently seen in spring and fall. Laryngitis is also frequently due to the inhalation of impure dust, or of otherwise irritating air. Alcohol contained in distillery mash probably has a similar effect, and cattle fattened on such mash frequently develop the symptoms of acute laryngitis (so-called distillery mash cough).

Chemicals and traumatic insults may likewise produce a noxious effect, as for instance in sudden and awkward drenching with fluid medicines or in the introduction of pills with a stick or the removal of foreign bodies wedged in the pharynx of cattle. The larynx is also irritated mechanically by long-continued convulsive cough or barking or bellowing.

A laryngitis may develop secondarily by the continuation of an inflammatory process of the nose, the pharynx, the trachea, the bronchi and of the lungs (catarrh, lungworm disease, pneumonia, etc.).

It is of importance that the secretions from the pharynx and from the deeper respiratory tract may come in contact with the larynx. Infection is frequently the cause of laryngitis in a number of infectious diseases (strangles, influenza, distemper, malignant catarrhal fever, tuberculosis, glanders, etc.).

**Chronic laryngitis** is produced by similar causes as those which are responsible for acute laryngitis, if these causes act

upon the mucosa for a longer time or repeatedly. Consequently chronic laryngitis is observed particularly among such animals as are kept permanently under unfavorable conditions, especially in impure air. In old dogs one frequently sees an obstinate chronic laryngitis.

Other causes are: disturbances of circulation in chronic heart disease, chronic inflammatory diseases of the deeper respiratory passages and of the neighboring organs, new formations (cysts, fibroma, sarcoma, carcinoma, or other tumor-like swellings—polyps, actinomycosis, tuberculosis, glanders), particularly those which have a tendency to ulcerate.

**Susceptibility.** Idiopathic laryngitis is seen particularly in pet animals with little power of resistance, for instance in horses kept in warm stables and in house dogs.

**Anatomical Changes.** In acute laryngitis the epiglottis, the arythenoid cartilages and the vocal cords are intensely reddened and swollen; in a later stage they are abundantly covered with mucus and sometimes infiltrated with blood. In severe cases there occur round superficial ulcers (Bruckmüller).

In chronic catarrh the swollen parts are more grayish in color; in consequence of the swelling of the mucous glands the surface is finely granular (laryngitis granulosa) or the epithelia which have proliferated here and there may form small nodules. The mucosa is thick and may in obstinate cases be set with villous excrescences; in horses the mucous glands may occasionally be changed into small cysts.

**Symptoms.** The principal and most common symptom of acute laryngitis is cough; it is at first short, dry and rough, later drawn and moist, always, however, more or less painful. In the presence of great tenderness it may be weaker. The animals are at short intervals subject to a tormenting cough, sometimes in the form of convulsive attacks. They stretch their necks forward and backward. Horses often paw and exhibit an anxious look. Dogs sometimes vomit after coughing. These attacks generally come on after the action of an external irritant on the larynx, especially after the animal has been led out of the barn, after a draft of cold air through the barn door, after the ingestion of cold water, or of dusty or hot feed, or after sudden or prolonged exercise, while animals standing or lying at rest only cough occasionally. The patients sometimes expel a tenacious mucus through the mouth or nose; such mucus, however, more usually adheres to the pharyngeal wall and is subsequently swallowed.

The increased sensibility of the laryngeal mucosa may be demonstrated objectively by pressure upon the arythenoid or thyroid cartilages; even mild pressure will likely produce repeated coughing and the animal will attempt to get

away from the hand of the examiner. In some cases the head is seen to be stretched out and bent sideways.

Difficulty of respiration exists only when the mucosa is swollen more markedly. In such cases one also hears whistling and rattling noises during inspiration. These symptoms may finally lead to the clinical picture of edema of the larynx. An increased laryngeal sound may, however, be heard on auscultation of the larynx even in such cases where there are no other noises which can be heard at a distance. One also hears frequently rattling over the larynx and the windpipe. In horses and dogs one may observe hoarseness and an indistinct noise.

Fever exists only in the initial stage of simple laryngeal catarrh. The elevation of temperature, however, as a rule, amounts only to a few decimals of one degree (C.°). Catarrh of the larynx occurring during the course of infectious diseases is associated with moderate or even high fever. A scanty, serous, mucous or mucopurulent nasal discharge exists only rarely; also a moderately acute swelling of the submaxillary and peripharyngeal lymph glands.

**Chronic laryngeal catarrh** either remains after an acute attack or develops from the start as a slow process. The symptoms are on the whole similar to those of the acute form. As a rule, however, the cough is less painful, rough, crowing and dry; rarely somewhat moist and accompanied by a peculiar explosive sound. The inspiration following cough is occasionally characterized by an audible noise. During the day cough is usually infrequent; the secretions which collect during the night cause long-continued attacks of cough in the morning. The sensibility of the larynx is likewise increased, but the animals do not try to get away from pressure as energetically as they do in acute laryngitis.

The respiration usually remains unchanged; exceptionally only is the cleft between the vocal cords narrowed to such an extent that respiration is difficult, even at rest, still more during exercise, and that it eventually becomes whistling. The respiratory difficulties are still greater if the catarrh has developed in connection with a neoplasm. Symptoms of fever are observed only in the presence of acute exacerbations.

**Course and Prognosis.** Primary acute catarrh of the larynx lasts from a few days to one to two weeks. The cough is at first rough and painful, but gradually it becomes more moist and less convulsive, and finally disappears entirely. The disease rarely assumes a chronic character and then persists like a primary chronic laryngitis for weeks and months. If the duration is very long the mucosa undergoes changes which exclude the possibility of complete recovery. The conditions under which the patients live have an important bearing on the course; in secondary laryngitis the nature of the primary



affection influences the course. If the larynx has once been diseased there is a tendency to relapses, and acute exacerbations are not rare in chronic catarrh.

**Treatment.** The most important measures to insure a speedy recovery are rest and protection against external irritation. The patients ought to be placed in a moderately warm place, with pure, quiet air. They ought to receive dust-free feed; if possible, green mashed food and bulbs. When the weather is favorable the open air is particularly beneficial in chronic catarrh. Animals which are only slightly sick may be used for light work if the weather is mild and if the patients are not exposed to the wind, to colds or to drenching.

If a tormenting cough is frequent the tenderness of the larynx should be lowered by narcotics. Indicated are: morphium ( $\mathcal{R}$  morph. mur. 0.10, aq. amygd. amar. 12.0 M. S. every 2 to 3 hours, ten to fifteen drops for dogs); also codeine ( $\mathcal{R}$  codein. phosphorici 0.60 aq. dest. 150.0 M. S. every 3 hours, 1 to 2 teaspoonfuls for dogs); heroine ( $\mathcal{R}$  heroini mur. 0.10 aq. dest. 150.0 M. t. sol. S. every six hours, 1 to 2 teaspoonfuls for dogs). Larger domestic animals should receive these drugs in the proper doses by subcutaneous injection; heroine causes great excitement in horses and occasionally a strong depression in dogs. The salts of bromine alone or in combination with codeine are sometimes indicated.

Priessnitz' compresses to the region of the larynx (every 2 to 4 hours) are very serviceable, both in acute and in chronic laryngitis. Warm packs are less effective and are indicated only in very acute cases. The inhalation of water vapors is of good effect in the incipient stage when the mucosa is comparatively dry and very irritable. Tenacious mucus may be made more liquid by the inhalation of finely divided mucosolvent remedies (1 to 3 per cent solution of common salt, sodium carbonate or bicarbonate). In the presence of an abundant thin fluid secretion, astringent and balsamic drugs are indicated, such as alum (1-30%), chloride of iron (0.1-0.3%), creolin (2-3%), creosote (0.5-1.0%), nitrate of silver (0.1-0.5%), oil of turpentine, tar (aqua picis 10%).

**Inhalations** are applied in the same manner as in nasal catarrh (see page 7). The inhalation of aqueous vapors or fluids which easily evaporate does not require any special apparatus, while the spraying of other fluids can only be accomplished by special devices. Ordinary sprays are not very serviceable; more to be recommended are Siegel's or Bullin's inhalers, the best is Wasmuth's apparatus, which, however, is generally only available in veterinary clinics, on account of its high price. Horses and cattle must be made to inhale through the mouth, which if necessary must be kept open by a speculum. Inhalations ought to be repeated every three or four hours and should be of ten to fifteen minutes duration. In the treatment of smaller animals one may employ tubs with boiling water covered by a perforated board; the patients are placed on the latter.

Only a very small fraction of a spray finds its way into the larynx of larger animals; hence, when treating them a Frick or Bayer-Kieselbach spray apparatus should be employed. These are easily introduced in horses; in cattle they can be introduced with some difficulty into the pharynx like a rhinolaryngoscope, and in this way permit of a more direct spraying of the larynx. If convulsive cough is present the solutions ought also to contain morphine up to  $\frac{1}{2}\%$  in amount.

The best results are to be expected in chronic laryngitis from painting the mucosa of the larynx or from the insufflation of powders, in cases in which the animals do not get too much excited during such manipulations. Indicated are: acidum tannicum or alum, argentum nitricum and saccharum album āā; cocaine, eucain, orthoform (of each 0.01 gm. with 0.2-0.3 sugar). The painting is best done with a 5% solution of argentum nitricum.

The powder is applied to smaller animals either with a long and properly curved brush or with a feather or by the aid of a Rauchfuss powder-blower. Painting may be done with a brush or with a sponge fastened to a small rod. The applications must be repeated daily.

In larger animals one may use a Frick or a Bayer spraying apparatus, and powder may be applied with a Neubarth powder-blower.

Intratracheal injection (Dieckerhoff) may be performed in larger, as well as in smaller, animals. Larger animals receive 30 to 40 cc.; smaller animals 4 to 5 cc. of fluid, which is rapidly injected with a needle immediately behind the larynx in a direction toward the pharynx. The following solutions are used: 0.5% solutions of alum, tannic acid or sulphate of zinc; 0.3% solution of acetate of lead; 0.1% solution of argentum nitricum; in large animals if the cough is very tormenting morph. mur. is indicated in a 1% solution (10-20 or 0.5 to 2.5 gm.).

Intratracheal injections are generally looked upon as without danger, but they may usually be dispensed with. In dogs with a short and thick neck this otherwise simple operation requires great dexterity. The inflammatory swelling which sometimes forms at the site of the injection must be treated with cold applications. In larger animals the injection may be performed with a Hauptner syringe or with a trochar; in smaller animals with an ordinary Pravaz syringe.

If the laryngitis is contagious in character, isolation of the healthy, or at least, of the sick animals, and disinfection of the stable or barn is indicated.

**Literature.** Dieckerhoff, W. f. Tk., 1886, 15; B. t. W., 1889, 187.—Freund, B. t. W., 1907, 575.—Pöschel, Ueb. d. Anw. d. Inhalation, Inaug. Diss., 1905 (Lit. on Inhalation).

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**Epizootic Laryngo-tracheitis of Horses.** A usually very contagious catarrh of the respiratory passages of horses has been known since 1888. It usually affects the mucosa of the larynx and trachea; at other times that of the bronchi, or again that of the respiratory parts situated anteriorly to the larynx. According to its variable location the affection is known as infectious laryngitis, infectious enzootic catarrh of the upper respiratory passages, enzootic cough, laryngo-trachéite épizootique (French), bronchite infectieuse, also frequently "la grippe." To this group probably also belongs the contagious pharyngitis of Dieckerhoff. According to Meyer "Skalma" (Dieckerhoff) is also a bronchial complication of this affection. Meyer claims quite properly that all these affections are only varieties of one and the same etiologic entity.



**Etiology.** In its epizootical and clinical respects the disease is very similar to equine influenza and cannot be separated from it completely the more so since the etiology of equine influenza is not yet cleared up. The possibility, however, of the occurrence of contagious catarrh independently of equine influenza cannot be denied, considering the fact that cattle likewise suffer from an epizootic catarrh of the respiratory passages and horses may suffer from epizootic laryngo-tracheitis, even after having previously had an attack of influenza. On the other hand, this is no conclusive proof since one attack of influenza confers immunity only for a short time.

The disease is usually observed annually. The extent of its prevalence, however, varies considerably in different years. Its very contagious nature is responsible for a frequently rapid extension within a few days among the horses of the same stable so that it assumes an enzootic and not uncommonly an epizootic character. The introduction of the disease occurs with newly acquired horses, with remounts, etc. The contagion may also be conveyed by persons or by the air. Younger horses are most susceptible. In some epidemics, however, horses are affected without reference to age. The period of incubation is 1 to 5 days according to past experiences.

**Symptoms.** The most prominent symptom, which is never absent, is a dry, short, strong, rough cough. If it is painful at all it is only moderately so. The cough occurs either very frequently or on the contrary after longer intervals. A marked tenderness of the larynx and of the trachea can be demonstrated. A serous, later on mucous, but usually scanty nasal discharge comes on which subsequently often becomes more abundant and lumpy. If this is the case the lymph glands of the laryngeal entrance swell moderately and mucous râles are heard over the trachea. In some cases bronchitic symptoms become manifest. The conjunctivae often appear normal or if there is an elevation of temperature they are markedly reddened, often diffusely rose red or yellowish-red or even dark red; sometimes, on the contrary, pale; according to Meyer edema is seen in the lower portions of the legs, the abdomen and the sheath of the penis.

Fever is often present. The temperature usually does not rise above 39° C., but it may not uncommonly go up to 41°. Even if occasionally fever is not present at rest, more intense exercise will elevate the temperature and at the same time accelerate the respiration and the pulse beat. Many patients show lassitude and malaise which contrasts with a comparatively insignificant psychic depression.

Concerning weakness or paralysis of the hind extremities (lumbar cord disease) following catarrh of the upper respiratory passages as observed by Kull and Duvinage, see enzootic paralysis of the cord.

The **course** of the disease is generally benign; the disturbances disappear in 8 to 10 days and after 8 or 10 days more the animals are able to work again. In some epidemics and in foals under two years old the period of convalescence lasts several, up to 6 to 8 weeks. Sometimes pneumonia or pleuritis comes on as a complication, especially if the animals have been worked hard during the disease. One attack of the disease does not appear to insure any lasting immunity.

The **treatment** consists more properly in feeding mashes, beets or a dust-free dry feed, sojourn in the open air during the favorable

season, even light work in the open. These measures are better than the administration of medicines. Sometimes change of location brings about a disappearance of the epizootic. Complications which may come on require proper special treatment. The stables must be thoroughly disinfected.

**Literature.** Bächstädt, Z. f. V., 1904, 429.—Christian, Z. f. Vk., 1901, 206.—Joly, Rec., 1888, 612.—Lignières, Bull., 1897, 496.—Meyer, Münch. T. W., 1909, 361.—Pr. Mil., Vb., 1899, 1908.—Schuhmager, A. L., 1909, 135.—Zorn, W. f. Tk., 1888, 249.

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**Epizootic Laryngo-tracheitis of Cattle.** In 1890 there was observed in some parts of Bavaria an acute catarrh of the respiratory passages in cattle which spread enzootically and even epizootically, and which was believed to stand in some relation to an influenza epidemic which was then very prevalent in man. More or less similar enzootics and epizootics have since been observed repeatedly, and they have been designated as epizootic laryngo-tracheitis (Zimmermann) or as influenza-like affections (Bräuer, Prietsch), or as infectious catarrh of the respiratory passages (Schmidt, Lewek). According to Lewek all these various affections are one and the same infectious disease, peculiar to cattle, which makes its appearance either sporadically or enzootically, particularly in south Germany. Cattle distemper (Janson) or influenza of cattle (Harms) deviates materially from the above affection, but they may be here considered, since their true etiology has not yet been cleared up.

The simultaneous appearance in Bavaria of this disease and of human influenza was undoubtedly only an accidental occurrence. Zimmermann failed to observe a simultaneous occurrence of the two diseases.

Epizootic laryngo-tracheitis or the influenza-like affections of cattle are contagious and are often imported by newly acquired animals (Zimmermann, Schmidt). The disease is evidently of bacterial origin. (In 8 cases examined Lewek found Gram-positive bacilli four times, once Gram-positive cocci and three times a mixture of both.) A longer transportation on railroads or aboard of ships seems to predispose to the infection and the disease is therefore often observed in cattle after shipment. According to Zimmermann the period of incubation is two to three, and according to Lewek, one to four days.

A contagious catarrh of the upper respiratory passages of epizootic character has also been observed in goats (Pr. Vb.).

**Symptoms.** We first observe a dry, painful cough coming on in attacks. The temperature rises to above 41° C., there is marked tenderness of the larynx and of the trachea, also marked reddening of the nasal mucosa and in the further course a watery or mucous nasal discharge associated with a mild conjunctivitis, occasionally also swelling of the eyelids. Rough, whistling, later on drawn and rattling, noises are heard over the region of the larynx. If bronchitis is present there is difficulty of respiration; the latter may also be due to a broncho-pneumonia which develops after two to three days in 50% of the cases (Schmidt). The appetite is usually much diminished. The disease usually lasts one to two, more rarely, two to three weeks, and

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ends, as a rule, in recovery. A fatal termination may occur in cases complicated with broncho-pneumonia.

In influenza-like affections of cattle one also observes the symptoms of gastric catarrh, arthritis and mastitis, to which may be added, in some cases, the signs of inflammation of the deeper structures of the eyes and a marked depression and lassitude on motion. Even in these cases, however, the course is usually benign.

**Treatment** is only indicated in those cases in which complications occur.

**Literature.** Bräuer, S. B., 1893, 117.—Janson, S. f. Tk., 1894, XX, 275.—Jb. bayr. Tzte., 1890.—Lewek, B. z. Kenntn. d. Erkr. d. Luftw. u. d. Lung. d. Rind. Diss. Dresden., 1909.—Markus, B. t. W., 1906, 655.—Pr. Vb., 1900, II, 9.—Prietsch, S. B., 1895, 96.—Schmidt, S. B., 1903.—Zimmermann, B. t. W., 1904, 167.

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**Japp Disease of Lambs.** Under this name Hasenkamp (D. t. W., 1909, 300) described a disease of lambs observed frequently in the provinces of Hanover and Westphalia in Germany. This disease only occurs while the animals are stabled and is characterized by an intense reddening and swelling of the mucosa of the larynx and trachea; by albumoid degeneration of the muscles of the larynx and by congestion of the lungs. Bacteriologic examination of the internal organs of animals dead from the disease has been negative. The affection has generally been looked on as mercurial poisoning after treatment of sheep-scab with a mixture of therosot. This view, however, is contested by Hasenkamp.

## 2. Croupous Laryngitis. Laryngitis crouposa.

(*Häutige Bräune, Halsbräune* [German]; *Laryngitis fibrinosa, angina membranacea, croup*.)

Croupous laryngitis is characterized anatomically by fibrinous membranous deposits on the laryngeal mucosa and is frequently associated with a similar affection of the trachea, occasionally also of the pharynx.

**Occurrence.** Croupous laryngitis is generally a rare disease. It is most commonly seen, if met with at all, among cattle and sheep; more rarely among horses, pigs and cats. It occurs occasionally in enzootic distribution. Guittard once saw an enzootic of croupous laryngitis among geese.

**Etiology.** Croupous laryngitis is primarily caused by the inhalation of hot air, smoke and other irritating substances (ammonia, chlorine gas, sulphurous acid, etc.). Cases which are occasionally observed after conflagrations or after disinfection of stables with irritating vapors are of such origin.

According to Roche-Lubin, the disease is also caused in some places by the practice of chasing sheep around for twenty-four hours in dusty places in order to increase the weight of the wool.

Aside from affections of the above nature, there is seen in cattle, sheep, horses, pigs, cats and young geese, a croupous

laryngitis due to an infection. Catching cold appears to play a predisposing rôle, because most of these cases are seen in spring and fall, on damp days or after sudden changes of temperature, also after a sojourn in the open during cold nights (according to Forneris often seen in the mountains of Piedmont). The microbe causing this infection is not as yet definitely known. Some observations, however, of Ernst seem to point to the bacillus necrophorus as the etiologic factor. This bacillus is frequently present upon the mucosae of herbivora and it may do harm if the resistance of a mucosa is lessened or if the bacterium has attained a higher degree of virulence. Other bacteria, perhaps streptococci, may have a similar effect.

The disease sometimes occurs secondarily in infectious diseases with similar or diphtheritic inflammations of the neighboring organs. Such diseases are: malignant catarrhal fever and croup of cattle, diphtheria of fowls and calves, rinderpest, sheep-pox, purpura hemorrhagica of horses, acute glanders, etc.

**Anatomical Changes.** Elongated, cylindrical or tubular fibrinous pseudomembranes are found at the base of the epiglottis, at the epiglottic-arythenoid bands, and also at the other portions of the larynx, and not uncommonly farther down in the trachea and even in the first portions of the bronchi. The membranes are 1-3 mm. thick in smaller animals; in larger animals considerably thicker grayish-white, or brownish in color. They are sometimes quite loose, almost liquid; at other times firmer and dryer. They are frequently partly detached, especially at the periphery, and still adherent in other portions. Under the pseudomembranes the mucosa is intensely reddened, sometimes containing extravasated blood, and in the deeper layers infiltrated serously or purulently. Sometimes the inflammation shows a diphtheritic character and deep losses of substance are then seen after the removal of the pseudomembranes.

The substance of the pseudomembranes presents an amorphous or reticular mass which contains white and some red blood corpuscles, also here and there epithelial cells in lumps, and various bacteria in the more superficial strata. The membranes swell up under the influence of acetic acid or ammonia, while they are dissolved by the caustic alkalies, lime water or lactic acid.

Post-mortem examination also shows other changes, namely, purulent bronchitis, catarrhal or croupous pneumonia, atelectasis of the lungs, acute swelling of the cervical and intra-thoracic lymph glands in cattle, also frequently croupous gastritis and enteritis. There are also usually signs of death from suffocation.

**Symptoms.** Croupous inflammation of the larynx begins with the symptoms of an acute laryngitis which increases



rapidly in intensity; the respiratory difficulty reaches a high degree within 1 to 2 days.

The temperature is considerably elevated from the start, at the same time the animals are much depressed, the appetite is suppressed, and rumination has ceased completely. Cough comes on soon; it is at first dry, short and strong; after a short time, however, it becomes peculiarly hoarse, convulsive and very painful. The respiration assumes a character pointing to stenosis of the larynx sometimes during the first day; the inspiration is markedly forced; the intercostal spaces and the lower portions of the thorax sink in strongly during inspiration. Each inspiratory act is accompanied by a peculiar whistling and rattling sound. Over the region of the larynx and the trachea stenosis sounds can generally be heard associated with moist râles and the hand placed over the larynx feels distinctly a laryngeal fremitus. The animals betray great anxiety. The nostrils become dilated to the utmost in horses; other animals keep their mouths open, or stretch out their tongues, hold their heads stretched forward, tremble, move their elbows away from their thorax, stand still with staring eyes; the mucosæ are cyanotic; the superficial veins are strongly congested; foamy, tenacious mucus is abundantly voided from the mouth. Increased laryngeal tenderness is permanently present, and even mild pressure brings about a convulsive cough; the difficulty in respiration may become so severe that it may lead to an attack of suffocation. In some cases the region of the larynx feels warmer and appears infiltrated with an edematous exudate.

During the attacks of cough the animals expel an abundant mucopurulent or purely purulent secretion through the nose and mouth. Toward the third to fifth day membranous shreds of fibrin are thrown out in addition or also longer fibrin cylinders, the caliber of which agrees with that of the larynx and trachea. The difficulties in respiration cease markedly directly after the expulsion of such masses and the general condition of the patient improves rapidly and may go on to complete recovery. In other cases the improvement is only temporary or amelioration and aggravation alternate repeatedly.

The neighboring lymph glands are swollen and tender; the pulse is rapid, small, and later on almost filiform. The appetite is poor; the feces are sometimes mucoid or mixed with shreds of fibrin. The secretion of milk ceases from the start.

**Course.** In a part of the cases laryngeal stenosis increases very rapidly and death occurs on the third and, in young geese, even during the first two days. Except in the last mentioned birds, the course is generally somewhat slower; does not, however, as a rule, extend over more than ten days. The end



comes usually in consequence of increasing laryngeal stenosis or in consequence of the lodgment of detached portions of pseudomembranes in the cleft between the vocal cords. A minority only of the affected animals recover.

**Diagnosis.** Croup of the larynx can only be diagnosticated definitely when the animals cough up shreds of pseudomembranes or void them during an attack of vomiting, or if the membranes can be seen directly in the larynx of the smaller animals. The laryngeal stenosis, however, which develops rapidly within one to two days with fever and in connection with a peculiar hoarse cough, usually furnish enough data to arrive at a correct diagnosis. In consequence of simple catarrh such marked stenosis does not develop while after the lodgment of foreign bodies or in acute edema of the glottis a high degree of stenosis is manifest within a few quarters of an hour. In stenosis due to neoplasm, there are no symptoms of an acute affection.

**Prognosis.** In small or younger animals the prognosis is very unfavorable, and recovery is the exception even in large and adult animals. In making a prognosis the general conditions of the animals should be considered; weak and otherwise diseased animals will hardly ever get over an attack. The expulsion of abundant masses of fibrin is a favorable symptom. The involvement of the lungs excludes recovery absolutely.

**Treatment.** The expulsion of the croupous membranes may be hastened in smaller animals by the use of emetics (sulphate of copper 0.05-0.5 gm., ipecacuanha 0.5-2.0 gm., tartar emetic, 0.1-0.3 gm.); if difficulties in deglutition exist these drugs are applied in the form of clysmata, or apomorphia may be given hypodermically (dogs 0.003-0.01 gm., cat 0.002-0.005 gm.) in aqueous solution. Hogs receive veratrine (0.02-0.03 gm. diluted in alcohol), likewise hypodermically. Vomiting must particularly be brought about in the presence of a sudden attack of suffocation, which is generally due to the lodgment of fragments of pseudomembranes in the rima glottidis. Detachment of the membranes is also favored by repeated inhalations, every one to two hours, of finely divided alkalis; lime water is principally used. If suffocation threatens tracheotomy must be performed. This operation may save the life of the animal whenever the inflammatory process is confined to the larynx or to the larynx and trachea, while the lungs are not affected. One may also try inunctions with mercurial ointment in the region of the larynx, moist warm or warm fomentations, excitantia (alcohol, wine) and the patients must sometimes be nourished artificially.

**Literature.** Anacker, Kochs Enzykl., II, 262.—Guitard, Pr. vet., 1902, II, 222.

### 3. Edema of the Larynx. Oedema glottidis.

By edema of the larynx, or oedema glottidis, is understood an accumulation of serous fluid in the loose submucous connective tissue of the epiglottic-arythenoid folds and of the ventricles of Morgagni, in consequence of which stenosis of the larynx is brought about.

**Etiology.** Edema of the larynx occurs as a primary affection after the inhalation of hot air, irritating acrid gases or dust-like substances; also after injury of the laryngeal mucosa; its development may be favored by preceding hard work, especially by excitement which has led to hyperemia. In this manner one may explain the occurrence of edema of the larynx after forced marches on dusty country roads (Buguiet) and at conflagrations. In young ducks fatal edema of the larynx is sometimes caused by the sting of bees which have fallen into the water when they were tired out and have been ingested by the young ducks.

In the overwhelming majority of cases, edema of the larynx is a secondary affection, coming on principally during severe disease of the larynx or of the organs in the immediate neighborhood of the former (pharynx, tongue, parotid gland, laryngeal and retropharyngeal lymph glands). As a collateral edema the affection is seen in certain infectious diseases (anthrax, hemorrhagic septicaemia of deers, of buffaloes, hogs and in hog cholera, petechial fever, pox, etc.); urticaria in cattle is sometimes associated with oedema glottidis (Wyssmann, Albrecht).

Congestion (chronic heart disease), compression of the jugular vein, traumatic pericarditis (Liebetanz) or cachexia may give rise to edema of the glottis; such cases are however rare when compared with various edemas in other places of the body.

The fat collecting in large amounts around the entrance to the larynx in fattened hogs produces, according to L. Weisz, an enlargement of the thyroid gland by compression of the veins, and the pressure of the thyroid upon the larynx causes a whistling sound (animals so affected are called "lung-whistlers.")

**Anatomical Changes.** The epiglottic-arythenoid folds, the lateral bands of the vocal cords, the folds between the epiglottis and the tongue, form trembling gelatinous ridges with either a reddened or a pale surface. These ridges collapse after an incision, and discharge a clear, more rarely turbid, serous fluid, and the mucosa then forms folds. Exceptionally the connective tissue of the ridges is found infiltrated with a purulent exudate, especially if the edema occurs in connection with deeper inflammation of neighboring parts (perichondritis, abscess formation). Simultaneously with oedema glottidis or independently

of it, an edematous infiltration of the submucosa is occasionally seen on the dorsal aspect of the trachea.

**Symptoms.** The development of inflammatory edema of the glottis takes place very rapidly; the symptoms of stenosis of the larynx appear within one quarter of an hour or within a few minutes. The animals are attacked by rapidly increasing difficulty in respiration; the inspiration especially is very forced and takes place with a marked sinking in of the intercostal spaces and of the other yielding structures of the thorax; it is accompanied by a whistling, rattling sound; expiration however is comparatively easy. The animals betray great anxiety, the eyes are staring, the eyeballs protrude, the mucosae become cyanotic, the superficial veins are enormously congested, the skin is covered with perspiration, the pulse is small and rapid. If the condition becomes worse, the animals usually succumb, generally after a preliminary fall in temperature and with convulsions.

In congestive edema the fully developed clinical picture is similar; however, its development takes several days and remissions are often noticeable.

**Diagnosis.** Croup of the larynx can be distinguished from primary acute edema glottidis because in the former case stenosis of the larynx occurs much more slowly, only after two to three days, and in connection with fever. Cough is markedly hoarse. Consecutive acute edema glottidis after a longer course of a primary disease leads either suddenly or gradually to stenosis of the larynx; the more chronic cases are distinguished from croup by the existence of the primary disease and by the absence of fever. Laryngeal stenosis due to lodgment of foreign bodies or pediculated neoplasms cannot be distinguished from edema of the glottis except by the history of the case; frequently the true nature of the affection is recognized only subsequently.

To avoid mistakes, the larynx should, in smaller animals, be inspected with the unaided eye, in horses, with the electric rhinolaryngoscope; in cattle, on the other hand, palpation of the larynx with the hand introduced through the pharynx is to be highly recommended, provided it can be done in the presence of the existing dyspnea.

**Treatment.** If there is danger of suffocation, tracheotomy should at once be performed, which will immediately alleviate the difficulties of respiration, and will be followed by recovery in primary cases or in cases with moderate inflammation, provided that there is no complicating edema of the trachea. Where, in smaller animals, the symptoms are not so threatening, one may try the ingestion of small pieces of ice, the inhalation of water vapors, or painting with astringents,



inunction with gray ointment. Nain succeeded in one case in causing the disappearance of edema of the glottis by repeatedly pulling out the tongue of the patient.

The primary disease must, of course, be properly treated.

**Literature.** Guenther, Z. f. Vk., 1908, 481.—Liebetanz, B. t. W., 1908, 732.—Nain, Rec., 1908, 369 (Revue).—Weisz, A. L., 1909, 489.

#### 4. Neoplasms of the Larynx. Tumores Laryngis.

**Occurrence.** Tumors of the larynx are comparatively rare among domestic animals; they are encountered most commonly in the larynx of horses and cattle. Cysts develop occasionally in horses, rarely in cattle, on the anterior surface of the epiglottis, exceptionally under the cricoid cartilage; they sometimes attain the size of a hen's egg and are composed of a wall of several layers surrounding a mucoid mass. Fibromata, lipomata, carcinomata, melanomata, lymphosarcomata, are exceedingly rare. Van den Eeckhout observed stenosis of the larynx in a horse due to an enchondrosis of the arytenoid cartilages. The round, pyriform or nodular hypertrophies of the mucosa (hyperplasia polyposa mucosae laryngis), occasionally seen in the course of chronic laryngitis, are also to be mentioned here.

Clinically similar to true neoplasms are tuberculous swellings seen not infrequently in cattle and exceptionally in dogs (Cadiot). These tuberculous masses may reach the size of a hen's egg; they are sometimes pediculated, generally situated behind the lower segment of the vocal cords; and when the process extends they may spread to the outside of the larynx. Actinomycomata develop generally between the base of the epiglottis and the rima glottidis and they spread over the surface.

**Symptoms.** Neoplasms arising from the epiglottis cause difficulties in respiration and deglutition; tumors developing in other parts of the larynx give rise to stenosis sounds and to difficulties in respiration. In either case occasional or periodic attacks of cough occur. Tumors of the epiglottis interfere with respiration, particularly during drinking, while other tumors of the larynx, unless pediculated, produce a gradually increasing dyspnea, which comes on during exercise only in the early stages. Pediculated tumors, on the contrary, produce attacks of dyspnea, since they are aspirated from time to time into the cleft between the vocal cords. According to the seat and the mobility of the neoplasm, either inspiration or expiration or both may be accompanied by difficulty and by a rattling or a whistling sound. The dyspnea which comes on in attacks, suddenly disappears or diminishes considerably; i. e., when the neoplasm becomes displaced out of the air



channel by a powerful attack of cough. Holterbach observed complete aphonia in a head of cattle with a tuberculous tumor in the larynx. Nasal discharge, sometimes streaked with blood, is not uncommonly present. Tumors situated in the anterior portion of the larynx in smaller animals can be immediately seen; in horses, however, the inspection of the larynx must be made with the rhinolaryngoscope, or they may be felt in horses and cattle with the hand introduced into the pharynx. (Horses must be laid down before manual exploration; a preliminary tracheotomy is indicated to avoid the danger of suffocation.) A swelling in the external aspect of the larynx is seen very rarely, and then only in laryngeal tuberculosis.

**Diagnosis.** The presence of tumors in the larynx can be ascertained beyond doubt only by an internal examination of the larynx; suspicion is, however, strongly aroused by the afebrile, chronic course, by dyspnea, coming on in attacks or gradually, with increasing intensity, occasionally accompanied by difficulties in deglutition, by stenosis sounds, by convulsive paroxysmal cough which sometimes relieves the dyspnea, and finally by non-tender swelling adherent to the skin on the outside of the larynx, with the signs of laryngeal stenosis. Sometimes it may be necessary to perform laryngotomy inter-cricothyroidea (Plósz).

**Treatment.** The difficulties of respiration can be relieved only by operative procedure. Pediculated neoplasms, or such at least as are not spread out over a large surface, may, after a preliminary tracheotomy, be removed with the écraseur introduced through the pharynx. (Almy removed cystic tumors of the epiglottis in this manner, in two horses.) The extirpation of flat, spreading neoplasms, or of those situated more posteriorly, requires laryngofissure. In actinomycotic tumors internal treatment with iodide of potash may be tried.

**Literature.** Holterbach, D. t. W., 1906, 541.—L. May, B. t. W., 1908, 176.—Mörkeberg, Z. f. Tm., 1907, XI, 63.

### SECTION III.

#### DISEASES OF THE BRONCHI.

##### 1. Bronchial Catarrh. Catarrhus Bronchialis.

(*Bronchitis Catarrhalis.*)

Bronchial catarrh consists in an inflammation extending to a variable extent into the tissue of the mucosa. It may either affect the larger bronchi (macrobronchitis) or only the finer bronchial branches (microbronchitis, s. bronchiolitis, s. bronchitis capillaris), or it may extend all over the bronchial tree (bronchitis diffusa). Catarrh of the larger bronchi is usually associated with catarrh of the trachea and larynx.

**Occurrence.** Bronchial catarrh is one of the most common affections of domestic animals and is seen in all kinds of animals, particularly in horses, dogs and cattle. Sometimes it occurs in horses, cattle and goats in epizootic form.

**Etiology.** Taking cold plays an important rôle in primary acute bronchial catarrh. Cold and wet weather, north and east winds, staying in the open air, pasturing during cold nights frequently give rise to colds, and therefore many animals are affected simultaneously in spring and fall. The detrimental influence of cold under these circumstances, and that of drafty air affects especially very young or very old, overheated animals or animals which have been robbed of their natural protection by shearing. Horses are therefore frequently affected after hard driving, dogs after hunting, sheep after shearing.

The inhalation of contaminated air is a further cause of bronchial catarrh. Especially dangerous in this respect are finely divided or gaseous bodies, because they can penetrate into the deeper parts of the bronchial tree. Lubenau's experiments have shown that vegetable dust can penetrate deeply and remain for a long time in the bronchial lumen. Metal dust is more apt to produce chronic catarrh. Spores of mould are inhaled during the ingestion of mouldy, musty or spoiled feed, and an acute catarrh may be produced in this manner.

Fluids or small solid bodies which may get into the air

passages, while eating, may likewise irritate the mucosa more or less. Non-acrid, non-corroding and pure fluids are less dangerous since they are rapidly absorbed by the mucosa, as was shown by intratracheal injections. However, dried particles are often contaminated with saprophytic and other bacteria and they cannot easily be removed from the bronchi. Since substances are often aspirated in difficult deglutition, pharyngitis, paralysis of the pharynx or unconsciousness, these conditions also often lead to bronchial catarrh; aspiration may also occur when fluids are poured in in an awkward manner. Larger foreign bodies (stones, ears of cereals, needles, etc.) may exceptionally get into the bronchi.

Bronchial catarrh undoubtedly also owes its origin to an infection, and it is then liable to assume an enzootic character. Under this type are to be mentioned the common contagious bronchial catarrhal affections seen in studs, among army horses, described under the designation of enzootic catarrh of the respiratory tract. Its true nature, however, is up to date not yet known (see page 43). The bacillus pyocyaneus frequently causes enzootic, purulent bronchial catarrh in young pigs and in cattle. Other forms of enzootic infectious bronchial catarrh however occur likewise in cattle and in goats. (Pr. Vb., Grimm, Martens, Lewek, Reisinger.) The bronchial catarrh observed by Schmidt in cattle in transit may likewise be due to an infection. (For further details see the chapter on catarrhal pneumonia.)

Secondary bronchial catarrh is quite common, particularly in connection with diseases of the lungs. Rarely, however, does inflammation of the larynx or trachea extend to the mucosa of the bronchi.

Finally bronchial catarrh is frequently seen as a part of the clinical picture of specific infectious diseases (strangles, hemorrhagic septicemia, smallpox, foot-and-mouth disease, distemper, etc.).

**Chronic bronchial catarrh** is caused by the same factors which bring about the acute form if the detrimental cause acts permanently or if it leads to repeated attacks of the acute form. However, even then the affection of the bronchi is more commonly secondary in nature. Every continuous disturbance of the respiratory mechanism or of the pulmonary circulation brings about chronic bronchial catarrh, because not only deficient ventilation of the air passages and the decomposition of mucus due to it but also stasis of blood will cause a disturbance of nutrition of the mucosa. Consequently a bronchial catarrh is almost always seen accompanying chronic pulmonary emphysema, chronic interstitial pneumonia, organic heart disease, and chronic infectious diseases affecting the lungs (tuberculosis, glanders, actinomycosis, etc.). Animal parasites may also produce chronic bronchial catarrh (see lungworm disease).

**Predisposition.** Particularly predisposed to bronchial catarrh are very young, or on the contrary, very old animals, pet animals, anemic or cachectic animals. These may contract the affection after minor external causes and their recovery is difficult to secure.

**Pathogenesis.** The mucosa of the bronchi possesses protective features which will prevent noxious effects of external influences within certain limits. Cough excited by an irritation of the mucosa and the epithelial ciliary movements which are directed outward favor the expulsion of particles and micro-organisms out of the trachea and bronchi which have gained access to the bronchial tree. The covering of the mucosa with mucus protects the latter against the immediate effect of substances which have gained access, while the numerous lymph follicles that are situated in the wall of the bronchi favor the destruction by phagocytosis of small elements which may have penetrated into the bronchial wall itself. The very smallest bronchi lack these protective structures; they are, however, sufficiently guarded by the larger bronchi situated anteriorly to them. The protective agencies become, however, insufficient if the noxious insult has been very intense, if the protective apparatus has been paralyzed, or if the detrimental stimulus travels along the blood current. Whenever the harmful agencies have gained the upper hand against the protective apparatus, the blood vessels in the mucosa become dilated, the latter swells and an increased production of mucus occurs, the substance of the mucosa becomes infiltrated with a sero-cellular exudate which also collects in the lumina of the bronchi. The substances acting as inflammatory irritants get in the further course into the pulmonary tissue with the lymph current, where they again excite an inflammatory process. If inflammation lasts for a longer time, it leads to an increase of connective tissue and to a reduction of elasticity of the bronchial wall. If the catarrh is confined to the larger bronchi, the bronchial air current is not influenced; on the other hand, there is considerable disturbance in bronchiolitis and there is the possibility of an extension of the process to the pulmonary parenchyma.

**Anatomical Changes.** In acute catarrh of the larger bronchi (macrobronchitis catarrh. ac.), we find the mucosa reddened uniformly or in patches, either everywhere or only in some branches, sometimes studded with small dark red extravasated blood, more or less swollen, loosened and covered with mucus or with a more purulent secretion. The size of the mucous glands is from that of a millet seed to that of a hemp seed and they discharge translucent drops of mucus upon pressure.

Catarrh of the finer bronchi (bronchitis capillaris, s. bronchiolitis, s. microbronchitis catarrh. ac.), can frequently be



recognized on autopsy by the fact that the lungs do not collapse well after opening the thorax, since the air cannot escape from the bronchioles which are filled with an exudate. Emphysema of the margins of the lungs (emphysema vicarians) has the same significance, and also the wedge-shaped, dark, sunken-in areas of the consistency of meat (atelectasia), while similar but firmer areas which protrude above the general pulmonary surface indicate an involvement of pulmonary tissue (broncho-pneumonia). Mucous or purulent drops containing more or less numerous fine air bubbles are seen on the cut surface.

In chronic catarrh, the bronchi likewise contain a serous, mucous or purulent, occasionally a caseous, exudate. The mucosa, however, appears grayish red or brownish, unequally thickened and sometimes, on the contrary, pale and thinned out. The connective tissue proliferation may in spots become so intense that villous excrescences are formed in varying degrees. Sometimes, tough nodules of the size of a millet or pea, are seen outside of the bronchial wall, but in connection with it, which may contain caseous material (peribronchitis nodosa). These processes may be accompanied by a proliferation of the interstitial pulmonary connective tissue, i. e., by an interstitial pneumonia.

In the deeper portions of the lungs, especially in the anterior and lower parts, bronchiectasias are formed occasionally which are variable in shape (b. cylindriformes, fusiformes, sacciformes, etc.), and which, in larger animals, may assume the size of a fist. They always contain an abundant dirty-grayish, mucopurulent secretion, which is occasionally fetid.

In aspiration-bronchitis, in the presence of bronchiectasias, occasionally also in simple chronic bronchial catarrh, the exudate and later on the bronchial wall itself decompose with the development of a dirty-greenish color and of a fetid smell (bronchitis putrida). According to the nature of the case, the bronchial glands sometimes show acute, sometimes chronic, swelling.

**Symptoms.** An invariable symptom of **acute catarrh of the larger bronchi** is cough. It is short, dry at the beginning, as long as there is no secretion in the bronchi, and later moist, with the increase and liquefaction of the secretion; then masses of secretion are expelled through the mouth or nose. Cough sometimes occurs in paroxysmal attacks. Respiratory difficulty occurs only whenever the process is more extensive, especially in the initial stage. Percussion of the thorax shows nothing abnormal; auscultation reveals various râles. At the very beginning, before any secretion whatever has occurred (bronchitis sicca), these râles are absent, and one may perhaps only hear a rough, vesicular breathing sound, but râles appear on the second or third day and remain present throughout the whole course of the disease. The râles are sharp and crackling

as long as the secretion is thick and tenacious; they become more dull, moist and more numerous after the secretion has become more abundant and more fluid. Coarse and dull râles suggest, to a certain degree, the involvement of the larger bronchi, while high fine râles speak for involvement of the small bronchi. Very loud strong noises indicate, in general, an affection of the more superficial parts, those which are less audible disease of the deeper portion. Sometimes the sounds are so pronounced that they can be heard in the vicinity of the patient without auscultation and the râles arising near the surfaces may be detected by the hands placed on the surfaces (*fremitus bronchialis*). Commonly also such other sounds as whistling, hissing, spinning are excited by the vibrations of lamellæ of tenacious masses of secretion.

Nasal discharge, which is often present, has generally a grayish-white mucous or mucopurulent appearance. It contains, at the beginning, few formed elements; in the further course, however, we find cell detritus, ciliated epithelia and many pus corpuscles.

The disease is generally initiated by a febrile elevation of temperature; enzootic cases which are due to an infection are particularly characterized by elevations reaching to 40° and 42° C. Sometimes a fall of temperature occurs as early as the second, generally on the third day. Increased pulse-beat, dullness and lassitude are likewise observed.

The symptoms of **acute catarrh of the finer bronchi** are usually preceded by the signs of macrobronchitis, the clinical picture of the former rarely coming on immediately; the cough, which is likewise always present, is weak at the start and may persist until the end, accompanied by little if any expectoration. The respiration is accelerated and forced especially during expiration; sometimes paroxysmal attacks of suffocation are observed.

The percussion sound is sometimes highly resonant toward the boundaries of the lungs, and in such cases the pulmonary boundary is usually displaced backward. This may also be so pronounced that the boundaries as mapped out by percussion may be in a line with the costal arches (observed by Marek in horses and dogs). Atelectatic portions of the lung rarely produce dulness on percussion, and whenever such is distinctly present it usually indicates that a bronchopneumonia has occurred.

Auscultation demonstrates fine râles (high pitched), sharp crackling sounds or crepitant râles, often in connection with other sounds. The vesicular breathing sound is accentuated in many places, in others less distinct or absent, in consequence of the permanent or temporary closure of some bronchial branches with masses of secretion.

Bronchiolitis is generally accompanied by fever lasting several days or persisting throughout the whole course of the

disease. The pulse is accelerated, there is dullness; lassitude and marked diminution of appetite are also always present.

**Chronic bronchial catarrh** develops in some instances from acute catarrh. As a rule, however, it arises very insidiously and develops slowly from the start. For a long time only cough is observed. It comes on at first during exercise or after the inhalation of cold air or after the effect of other irritating factors. Generally drawn and moist, rarely painful, it appears occasionally in longer paroxysms, eventually with the expulsion of a thick mucous or mucopurulent secretion. The respiration is not interfered with in the beginning stage; later on, however, it becomes forced, and work or rapid motion bring about sneezing, which in older animals is so frequently caused by chronic bronchial catarrh.

Auscultation gives the same sounds as in acute catarrh; although dry râles and especially purring and whistling sounds are particularly prominent. Fever is absent aside from the not uncommon cases with acute exacerbations and from cases of a secondary nature where the primary affection is accompanied by fever. The general condition sometimes remains undisturbed for a long time; but if the catarrh exists for several months emaciation and anemia are seen, particularly in older animals.

The bronchial catarrh which is frequently seen in cattle, and which is probably due to an infection with the bacillus pyocyaneus differs from the above-described picture only in this respect that the wheezing, whistling, and purring sounds and crepitation, are best heard at the portions of the lung covered by the shoulders. One can also demonstrate tenderness of the anterior intercostal spaces. The respiration is variably affected according to the duration and is either forced or accelerated. The general condition and the production of milk remain undisturbed. In some cases, bronchial catarrh is followed by bronchopneumonia (see there).

For details about bronchitis scleroticans of horses, as described by Grüter, see in the chapter on chronic interstitial pneumonia.

**Putrid bronchial catarrh** is characterized by a peculiar, disagreeable, sweetish smell of the exhaled air, or of the occasional nasal discharge which is particularly abundant after paroxysms of coughing, and by a dirty discoloration of the nasal secretion. Otherwise the symptoms are the same as in other forms of bronchitis; however, gangrene of the lungs frequently follows upon putrid bronchitis. In very rare cases one is able to obtain tympanic sounds on percussion, in some part of the lower thoracic region bronchial or amphoric respiratory sounds, or possibly metallic râles as signs of an existing bronchiectasia.

**Course.** Acute catarrh of the large bronchi generally runs a course of two to three weeks; but occasionally recovery takes



place after a few days. Bronchiolitis, however, is characterized by a slower course and is frequently accompanied by bronchopneumonia, which not uncommonly leads to a fatal issue; bronchiolitis, however, may alone be the cause of death.

Chronic catarrh always lasts several weeks and may last a number of months and even years. This depends upon whether the catarrh is a primary or a secondary affection, and under what conditions the patient lives. In protracted cases, acute exacerbations are occasionally seen, also attacks of suffocation, and these may lead to a great impoverishment of the condition of the patient.

**Diagnosis.** The diagnosis of bronchial catarrh meets with no difficulties, the presence of râles or of whistling or purring sounds being characteristic. One also hears râles in edema of the lungs and in pulmonary hemorrhage, but these affections come on suddenly with very grave symptoms and generally with a foamy or hemorrhagic nasal secretion. The character of the râles and the degree of respiratory difficulty point to the affected bronchi, while the onset and course of the disease indicate its acute or chronic character. It is, however, sometimes difficult to determine whether a bronchial catarrh is primary or secondary in character. Only a very careful examination of all organs can secure against error. The absence of symptoms pointing to any other disease speaks in favor of a primary bronchial catarrh, and the absence of metallic râles, aside from the rare cases of bronchiectasias, as well as the generally favorable course, likewise speak in favor of primary catarrh, while high fever and great difficulty in respiration point to an affection of the lungs. However, catarrh of the finest bronchioles also leads to symptoms similar to those last mentioned, so that it is sometimes difficult to decide whether we are dealing with a simple bronchial catarrh or whether catarrhal pneumonia is already present.

**Prognosis.** Idiopathic catarrh of the larger bronchi is in general a benign affection. Catarrh of the finer bronchi, however, may in itself lead to suffocation, or it may become complicated with catarrhal pneumonia. Since experience has shown that this form of catarrh preferably affects very young or very old and debilitated animals, the prognosis in such animals is always dubious and is decidedly unfavorable in the presence of intense dyspnea and high fever. Diffuse bronchitis, which is seen after conflagrations, is likewise of very grave significance. Enzootic bronchial catarrh usually takes a favorable course and only comparatively rarely leads to death of the animals, usually after a complicating pneumonia or pleuritis has developed; even these latter complications do not exclude the possibility of recovery. In enzootic bronchial catarrh of cattle of commerce Schmidt observed a mortality of 30%.



The prognosis of secondary bronchial catarrh depends upon the nature of the primary disease.

**Treatment.** Rest and pure air are the most potent factors in securing speedy relief from bronchial catarrh. Hence the patients must be kept in uniformly warm, clean places, the barns must be well aired and the sojourn in the open, in sunny places free from draft, is to be favored. Larger animals should not be used for work during the whole duration of an attack of bronchial catarrh, even if it is confined to the larger bronchi. The feed should consist of easily digestible, dust-free material containing an abundance of water; as a drink the patients should have pure, if possible slightly alkaline, not too cold water. Frequent and tormenting cough, particularly in the initial stage, should be ameliorated by the same narcotics which are recommended for laryngitis (see page 42). In the further course, after the secretion has become more abundant and more liquid, its expulsion must not be prevented by a suppression of the cough.

In dry bronchitis sojourn in moist air exerts a favorable influence and this treatment is particularly serviceable in the case of smaller house animals which are usually kept in rooms. The contents of the air in water vapor can be increased by placing flat vessels filled with water near the stove or radiator or by the use of a spraying apparatus. The object of frequent inhalations is the same. The benefits derived from them must, however, not be overestimated, because it has been shown experimentally that vapors and finely divided fluids are deposited already in the nose and pharynx, so that only a small part of them can get into the deeper respiratory passages. For inhalation purposes the same substances may be used which were recommended against laryngitis (see page 42). If the exhaled air has a fetid odor, one may use a finely divided spray of corrosive sublimate (1:2000); 2-3% solution of creolin; 1-2% solution of carbolic acid. In intense dyspnea due to an extensive bronchiolitis the administration of oxygen may become necessary.

The intratracheal injection of drugs can only have an insignificant place in practice. Aside from the fact that the methodical carrying out of such treatment meets with difficulties the ingested fluids (as shown by Bärner) only get into the larger bronchi of the anterior pulmonary portions and the watery solutions are there absorbed too rapidly. A certain success might be expected after the injection of larger masses of fluids or after oil in emulsions. The following are adapted for intratracheal injections: sodium bicarbonate (2-3%); zinc sulphurate (1½%); argentum nitrate (0.1%), also Lugol's solution (1:5: 100-200), and oil of turpentine (with olive oil āā). Larger animals receive from 20-40 gm. of the watery solutions; smaller animals 2-3 gm. at one dose injected into the trachea;



of the oil mixtures, of each 15-20 or 1.0 gm. Masson & Vazeux have used creosote with good success in putrid bronchial catarrh (creosote 1.0, alcohol 40, aqua 40, of this mixture 20 cc. twice daily). A 4% formalin solution might be used instead of the creosote; sometimes, however, the former produces a prolonged irritation, which causes cough. To each one of these solutions 1-2% tincture of opium might be added in order to lessen the irritability of the mucosa.

The value of internal medication is very doubtful. Expectorants and solvents of the mucus are in general use, for large animals in the form of pills and electuaries, for small animals in the shape of solutions. Such drugs are: stibium sulf. aurantiacum (10-20 or 0.5-1.0 pro dosi), ammonium chloratum (8-15 or 0.2-1.0 gm.), root of senega (in a decoction 10:15 for dogs) and root of ipecacuanha (0.5:15.0 in tablespoonful doses), etc., 2-3% of liquor ammonii anisatus is added for smaller animals. In a horse with bronchiolitis, Hermann had good results by the repeated subcutaneous injection of iodipin (40-50 gm.). When there is abundant secretion of mucus, especially in the smaller bronchi, and difficulty in respiration depending on it, emetics are indicated in smaller animals, since vomiting raises the pressure and produces a stronger air current in the bronchi, which favors the expulsion of masses of secretion. Such drugs are: apomorphine hydrochlorate (0.005-0.01:50.0, combined with 0.05 gm. morph. hydrochlor.; every three hours a tablespoonful for dogs); tartarus stibiatus (1:100.0 every fifteen minutes one tablespoonful); ipecacuanha (1-20:150 aqua), etc. Atropine (horses and cattle 0.03-0.05 gm., dogs, 0.002-0.01 gm., subcutaneously) has a tendency to suppress excessive secretion and to relieve dyspnea for some time. Priessnitz's applications may be made to the chest.

The general condition must be watched in very young and in very old animals and stimulants must be given in the presence of debility (camphor, ether, alcohol, caffeine). Larger animals must be rubbed down two to three times a day.

**Literature.** Bärner, A. f. Tk., 1899, XXV, 67.—Behrens, D. t. W., 1904, 62.—Berger, Z. f. Infkr., 1907, III, 101.—Grimm, S. B., 1888, 69.—Grüter, Beitr. z. Kenntnis d. Bronchitis chr. d. Pferdes, Diss. Zürich, 1909 (Lit.).—H. Holt, Z. f. Infkr., 1907, III, 155.—Lewek, Beitr. z. Kenntnis d. Erkrank. d. Luftwege und d. Lungen d. Rindes, Diss. Dresden, 1909 (Lit.).—Lubenau, A. f. Hyg., 1907, LXIII, 391.—Martens, B. t. W., 1906, 655.—Petropawlowsky, A. f. Vet.-Wiss., 1906, 14.—Pr. Vb., 1900, II, 9.—Reisinger, Monh., 1908, XIX, 193 (Lit.).—Schmidt, S. B., 1903, 79.

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**Bronchial Croup** (*Bronchitis crouposa*). Cattle and sheep suffer from a croupous inflammation of the bronchial mucosa under the same conditions which bring about croup of the larynx, or it may be secondary to nasal or laryngeal croup. In idiopathic bronchitis crouposa the mucosa of all bronchi or of the majority of them is covered by tubular masses of fibrin. These masses interfere more

or less with the exchange of gases and they close the finer bronchi completely.

Hence the symptoms of this affection consist in a very grave dyspnea which reaches a high degree, either suddenly or more rarely after a preliminary fever which is not well marked. One hears rattling, whistling and purring sounds over the thorax. They are as a rule very loud and may be felt as a pectoral fremitus. The disease is recognized in its true nature only after the patients have coughed up croupous membranes, the source of which is made obvious by their tubular shape and branching arrangement. This does not, however, occur in all cases, because the animal may suffocate without any expulsion of membranes or the latter may not be thrown out but may be swallowed and so escape notice.

The disease may reach its full development within 2 to 3 days and may then lead to suffocation, especially in young animals, while older and stronger animals may occasionally recover after the expulsion of the membranes.

The treatment is similar to that recommended for laryngeal croup; tracheotomy, however, is of no avail, and the chances of successful treatment are not as good since we cannot reach the deeper portions of the respiratory tract.

[The disease of cattle and sheep described above is similar to one occurring in man and now generally called bronchitis fibrinosa. It is, however, a very chronic affection in man, which usually leads to a fatal issue. Translator's note.]

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**Bronchial Catarrh of Fowl.** This disease manifests itself in cough and accelerated respiration, accompanied by râles formed in the trachea and audible at a distance; on flying the râles may be increased to a rattling sound. Very marked difficulties in respiration point to an affection of the pulmonary parenchyma.

Internal treatment is not promising. Zürn recommends the administration of the following: Ammon. chlorat. 0.5; mel. 0.5; aqua foenic. 50.0 (table or tea spoonful doses 3 to 4 times daily). More successful is the inhalation of hot water vapors, or of a fine spray of mucosolvents (1% sol. common salt, sod. carbonat. or bicarbonat.). These are employed in such a manner that the opened bill of the bird is held over a vessel with hot water or over an inhalation apparatus. Birds kept in the room may be treated so that a vessel with hot water is placed next to the cage, while both are covered with a cloth. Sprays must be directed into the cage. Care must be taken that the birds get enough air for breathing.

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**Contagious Inflammation of the Air Sac in Geese.** This is a general infectious disease which has been observed in epizootics among geese imported from Russia. It is characterized anatomically by a fibrinous inflammation of the air sacs and of the serous membranes.

The cause of the affection is, according to Bugge, a slender bacillus of about the same morphology as the bacillus of hog erysipelas. It does not, however, form colonies on ordinary nutrient agar like the bacillus avisepticus.

The most marked anatomical changes are found in the air sacs, which appear yellow in color while their internal surface is covered with thick, leathery, whitish-yellow masses. Similar deposits are found on the serous membranes of the liver, spleen, intestines, peritoneum



and kidneys. In consequence of the frequent simultaneous involvement of the peripheral bronchi nodules of pin-head to pea size or branching masses are found in the lungs.

The most essential symptoms are accelerated respiration with opening of the bill, with snoring sounds, also staggering in walking, difficulty in rising with kicking of the legs toward the head. The depression, which is present from the start, increases rapidly and the birds generally die within 8 to 10 days, occasionally also earlier and quite unexpectedly. The disease may be distinguished from fowl cholera because it occurs exclusively in geese, by the absence of hemorrhages and the presence of fibrinous deposits in the air sacs and by the microscopic detection of a slender bacillus which does not take a bipolar stain. In croupous laryngitis of young geese intense dyspnea is present from the start and the pseudomembranes are found in the larynx.

Since treatment is unpromising the disease can only be met by prophylactic measures (see fowl cholera).—(Bugge, Z. f. Infks., 1907, iii, 470.)

## 2. Narrowing of the Large Bronchi. Bronchostenosis.

**Etiology.** Stenosis of the larger bronchi may be caused especially by the accidental introduction of foreign bodies, ears of grain, fir-cones, pieces of bone, cartilage, wood, needles, etc., as they have been found repeatedly in the air passages of horses, cattle, swine and carnivora. (Szabo found a mouse in the air passages of a hog that had suffocated suddenly, which got there accidentally while the animal had been rooting.) Other causes are narrowing of the bronchial lumen by croupous pseudomembranes or compression of the bronchi by enlarged or suppurating bronchial or mediastinal lymph glands, aneurysms of the aorta, tumors of the lungs or of the mediastinum.

**Symptoms.** Narrowing or complete obturation of a larger bronchus diminishes the exchange of gases in the corresponding portion of the lung or prevents it entirely and causes difficulties in respiration, which come on suddenly in the case of the introduction of a foreign body, but more gradually in case of compression by a tumor. Particularly the former condition leads to convulsive paroxysms of cough. Difficulty of respiration is generally of a much graver form in consequence of sudden closure than it is in a stenosis which has developed gradually. The stenosis is usually unilateral, it lessens the excursions of the corresponding half of the thorax and causes a sinking in of the yielding portions of the thoracic wall in inspiration. A stenosis sound similar to that heard in laryngeal stenosis may likewise be observable.

The percussion sound remains unchanged as long as there is not a complete obstruction. The vesicular breathing sounds are absent over the affected half of the thorax, or at least weakened, and are intensified over the other portions of the chest. Over the affected parts are heard occasionally rattling or whistling sounds.



In stenosis caused by a foreign body the exhaled air later on becomes fetid and the temperature rises. In stenosis caused by compression one occasionally also observes edematous infiltration on account of the simultaneous compression of larger venous trunks, paralysis of the larynx due to compression of the inferior laryngeal nerve, disturbances of deglutition due to compression of the esophagus, and in ruminants chronic bloating.

**Treatment.** Since successful treatment is impossible, the animals ought to be utilized by early slaughtering.

### 3. Asthma bronchiale.

(*Asthma spasmodicum*, *Asthma nervosum*.)

Under this name difficulties of respiration are designated in human medicine which come on periodically in violent attacks, being caused by transitory impediments in the bronchioles, and are followed by a return to regular and free respiration. The absence of anatomical changes which might explain the respiratory difficulties points to a nervous origin of the affection. It is looked upon as a neurosis of the vagus and it may have a variety of underlying causes. Irritation of the vagus (*pneumogastricus*) may be caused by compression (neoplasms, enlarged lymphatics) or by bronchial catarrh or also by reflex irritation in morbid irritability of the nasal or pharyngeal mucosa (transmitted through the trigeminal nerve), or in certain anomalies of the abdominal organs, particularly the stomach and intestines, especially in a catarrhal condition of those organs (*asthma dyspepticum*). Following upon irritation to the vagus a tenacious secretion is found in the bronchial tree. The smooth muscle fibers of the latter contract convulsively. The bronchial lumen is narrowed, the entrance of air into the alveoli is interfered with and the attack of asthma is brought about. A tetanic contraction of the diaphragm occurs simultaneously, either by reflex irritation through the pneumogastric nerve or in consequence of the increase of carbon-dioxid in the blood. Inspiration and expiration are both interfered with during the attack. Signs of acute atelectasis of the lungs are also present and whistling and purring may be heard over the thorax.

Some veterinary authorities have reported similar cases, the identity of which, with asthma bronchiale, however, cannot be considered as proved, because it had not been considered that transitory stenosis of the air passages may occur and also that severe attacks of dyspnea sometimes occur in the course of permanent stenosis, in emphysema of the lung, in chronic uremia (Liénaux) and in chronic cardiac disease.

Grebe observed the following symptoms in the case of a horse: During the last ten days the animal had been breathing very heavily at each meal, and this condition lasted for one-half to one hour after the ingestion of food. During the attack the animal betrayed great anxiety, was sweating at the head and neck, the mucosae were somewhat cyanotic, the markedly difficult inspiration was accompanied by a strong dilatation of the nostrils and by low whistling sounds; the expiration was easy and free; respiration 28 per minute. The abdomen was

somewhat bloated; defecation retarded. During the examination the symptoms increased to such an extent that the animal threatened to fall down. During the intervals between the attacks neither at rest nor at exercise did any disturbances occur. All symptoms disappeared after an injection of eserine (Grebe thought that this case was identical with the asthma dyspepticum of man).

Penberthy reported six cases in which the attacks came on suddenly and disappeared just as suddenly after 2 to 24 hours, after the patients had expelled a bronchial secretion by a violent attack of cough. Improvement did not occur, however, and two of the asthmatic horses presented the signs of emphysema alveolare genuinum (!).—Boer saw the affection in two young cows which, in slight narcosis produced on inspiration a sharp whistling or hissing sound during the attack. The presence of pediculated tumors (tuberculous) in the larynx or its neighborhood had not been entirely excluded in these cases.

**Literature.** Boer, Holl, Z., 1888, XV, 23.—Grebe, A. f. Tk., 1889, XV, 255.—Penberthy, J. comp. Path., 1894, 359.

#### 4. Lungworm Disease. Bronchitis et Bronchopneumonia verminosa.

(*Verminous bronchitis; Verminöse Lungenphthise, Strongyluskrankheit, Lungenwurmseuche, Lungenwurmhusten* [German]; *Strongylosis pulmonum; bronchite et bronchopneumonie vermineuse, Strongylose bronchopulmonaire* [French].)

Lungworm disease is an affection which is usually enzootic, even epizootic. It occurs in the form of a bronchitis or bronchopneumonia of variable extent and is caused by lung palisade worms.

**Historical.** Epizootics among animals caused by worms were first described in the middle of the eighteenth century. They attracted more universal attention, however, only after another hundred years. The following have especially studied this affection: Davaine (1860), A. Koch (1883), Neumann (1888), A. Müller (1889), Tapken (1891), M. Schlegel (1899), Jeanmaire (1900), Doctor (1907), Joest (1908) and others.

**Occurrence.** The disease occurs in all countries, usually among animals which are kept in marshy pastures or in pastures which are frequently exposed to inundations or situated in lowlands. The disease sometimes occurs during stable feeding, especially in hogs, more rarely in cattle. Among domestic animals, sheep and goats are most commonly affected, in some neighborhoods also swine; more rarely affected are cattle and camels; horses, asses, cats, dogs and rabbits only exceptionally. Lungworms are very frequently found among some wild mammals such as deers, roes, chamois, boars and hares. As a herd disease, it is generally only met with in sheep and goats, especially among younger animals, and from time to time it causes

serious losses, particularly after wet summers. The enzootic appearance among swine herds is rarer; it is still less common among grown cattle and calves. Enzootics of the disease have occasionally been observed among camels. The disease appears not infrequently as a very destructive epizootic among the wild animals mentioned above.

According to Carnet half of all sheep and one-third of the cattle in Morocco succumb in some years. The disease occurs epizootically in South Oranais, according to Ben Danou, and is called "Reuch" by the natives, and when disease of the lung is simultaneously present they call the affection "Reuch-el-riya." Three-fourths of all sheep (many millions of animals) succumbed in Buenos Ayres from 1883 to 1886. The disease is quite destructive in some parts of Germany (on the lowlands of the Weser and the Jade rivers) among cattle and calves (Tapken). The disease is also widespread among swine in some provinces in Russia, and of the swine slaughtered 14% were found affected in St. Petersburg, 21% in Moscow and 48% in Riga (Schultz). Heavy losses among sheep were caused in Hungary in the year 1889.

In slaughtered animals lungworms are found frequently which have, however, not affected the health of such hosts unfavorably. Hertwig gives the following figures for the Berlin slaughtering plant for 1887-88. Among 275,049 sheep, 788 or 3%; among 419,848 swine, 3,237 or 7%. In the Budapest stock yards there were found from 1889 to 1903, among 90,883 sheep, 3,082 or 3.4%; and from 1902 to 1903, among 474,401 hogs, 320 or 0.067%.

**Etiology.** The palisade worms (*Strongyli Müller*) belonging to the filiform worms (nematodes) are usually long, slender, smooth, whitish worms, with a mouth-end at the anterior portion of the body. It is either round and smooth or surrounded by warts. The posterior portion of the male, which is always shorter, ends in a bursa which is variable in shape and which serves for grasping the female, being provided with two spiculi. The posterior end of the female is straight. The genital pore is sometimes behind, sometimes in the middle of the body.

The following palisade worms have been found in the respiratory organs of domestic animals.

1. ***Strongylus filaria Rudolphi*.** The anterior of the body somewhat smaller, mouth roundish without papillae; male 3-8 cm. long, bursa bent inward, supported by ten ribs; female 5-10 cm. long, posterior end pointed; the genital pore somewhat behind the mid-line of the body. Ova (Fig. 5) are oval, have a delicate hyaline membrane and contain well developed embryos within the mother animal.

The worm is parasitic in the respiratory organs of sheep and goats, also in roes, in gazelles, in camels, deer and fallow-deer.

2. ***Strongylus commutatus* Diesing** (*Str. rufescens*, *retortaeformis*, *Trichosoma leporis pulmonale*, *Filaria terminalis*). Filiform worm with rounded and flattened end, brown-red in consequence of the intestinal tract shining through. Male 1.8-3 cm. long, bursa very small and rounded; female 2.8-5 cm. long, genital pore immediately before the anus. Oviparous. Ova elongated oval, enclosed in a very delicate shell, do not show any segmentation in the body of the female.

The worm occurs in sheep, goats, rabbits, hares, roes and chamois.



3. **Strongylus micrurus** Mehlis. Long slender worm with pointed ends, smooth head and mouth. Male 3.5-4 cm. long with small simple bursa which is supported by five ribs and which is supplied with powerful spiculi; female 6-8 cm. long, posterior end strongly pointed, genital pore directly behind the mid-portion of the body. Ova (Fig. 6) are oval with delicate shell, containing an embryo in the mother animal. The head end of the embryo is thicker, the posterior portion of the body is S shaped.

The parasite occurs in cattle, fallow-deer, stags, exceptionally also in horses and asses.

4. **Strongylus paradoxus** Mehlis (*Str. longevaginatus*). Comparatively short whitish or brown worm with conical head and a mouth-opening surrounded by six small papillae. Male 1.2-2.5 cm. long, bursae composed of two wings which are supported by five ribs, spicula fine and long. Female 2.5-4 cm.; its pointed body end is curved, genital pore in front of the anus. It deposits ova and also live embryos. Ova (Fig. 7) short oval with a thick shell. The posterior end of the curved embryo is thickened in a globular manner. Occurs in domestic and wild pigs, exceptionally in sheep, roes and deer.

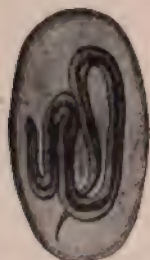


Fig. 5. Ovum of *Strongylus filaria*.



Fig. 6. Ovum of *Strongylus micrurus*.



Fig. 7. Ovum of *Strongylus paradoxus*.

5. **Strongylus Arnfieldii** Cobbold. White filiform worm; male 2.8-3.6 cm. long; female 4.3-5.5 cm. long. Ova elliptical, embryo with a fine caudal appendix. Occurs in horses and asses.

6. **Strongylus capillaris** Schlegel (*Nematoideum ovis pulmonale*, *Pseudalius ovis pulm.* Koch). A worm as thin as the threads of a spider web, cylindrical, brownish (hence called lung hair-worm); the body terminates behind in a point and is twisted like a corkscrew in the male. Mouth opening surrounded by four papillae. Male 1.4 cm. long. The ventral branch of its spicula is divided dichotomously and serrated; bursa, however, very small. Female 1-2.3 cm. long. Oviparous. (Railliet and Neumann believe this worm to be an undeveloped form of *Strong. commutatus* (*rufescens*); Schlegel has shown that it belongs to the Strongylidae. The mature worm penetrates into the pulmonary tissue after having deposited its ova; it becomes encysted there and it finally dies. The worm has been found in sheep, goats, roes and chamois.

7. **Strongylus sagittatus** Müller in stags.

8. **Strongylus pusillus** Müller in cats.

9. **Strongylus minutissimus** Mégnin in sheep (very rare).

10. **Strongylus pulmonalis** Ereolani in calves.



11. **Strongylus canis bronchialis** Osler (*Filaria tracheobronchialis*). This worm, the zoological position of which has not yet been definitely decided, is sometimes found in wart-like nodules of the trachea or larger bronchi in dogs.

12. **Strongylus vasorum** Baillet. The ova and embryos of this worm are found in the lungs of dogs (found heretofore in the neighborhood of Toulouse, France, and in Italy).

In the respiratory passages or in the lungs of cats are found exceptionally *Trichosoma aerophilum* Creplin, also the embryos of *Ollulanus tricuspis* Leuckart. The individual species of domestic animals may therefore contain several species of strongylus, viz.:

In sheep: *Str. filaria*, *commutatus*, *rufescens*, *capillaris*.

In hogs: *Str. paradoxus*.

In cattle: *Str. micrurus* and *pulmonalis*.

In horses and asses: *Str. micrurus* and *Arnfieldii*.

In dogs: *Str. canis bronchialis* and *Str. vasorum*.

In cats: *Str. pusillus*.

In rabbits: *Str. commutatus*.

As to the frequency in occurrence of individual species of lungworms in domestic animals which harbor several species of strongylus, the following may be said: In sheep one finds, most commonly, according to the general view, *Strongylus capillaris* and exceptionally only *Str. paradoxus* or *Str. minutissimus*; not uncommonly, however, there may be more than one, and in fact all of these three species of strongylus in one and the same host animal. It is said that the disease generally takes an enzootic character if the *Str. filaria* is present, more rarely if *Str. capillaris*, and still more rarely if other species of strongylus are present. Some observations, however, point to the fact that these conditions may be changed materially, particularly in some parts. Ranke found *Str. capillaris* in all sheep marketed in London. Ströse found this same worm in 66%, and Schlegel in 22% of the sick sheep, while the last-named author found *Str. commutatus* in 33% and *Str. filaria* in only 4%. It is also believed that goats are infected most commonly with *Str. filaria*; but Schlegel found the *Str. capillaris* in 92% of sick goats.

The development of lungworms is only partially known and in some species totally unknown. The worms deposit their ova in the air passages of the host, whereupon the embryos are either set free immediately, or, in the oviparous species, only after some time; and they are either expelled directly with the bronchial secretion into the outside world or they get into the pharynx, are swallowed and voided with the feces. Observations made on the embryos of *Str. filaria* have shown that, provided the temperature and moisture are favorable, the larvae which have arrived in the outside world go through several castings in moist soil, in pools of water, in marshes or in wet grass (casting may occur even in the air passages of the host in *Str. capillaris*); and they live then, according to some authors (Leuckart, Railliet) for some time in an intermediary host (insect, mollusc, angle worm). Only after such development will the parasites be able to invade the air passages of certain animals and there reach sexual maturity.

According to Gerlach the period of development to sexual maturity lasts at least eight weeks; however, the observation of Schlegel, that lungworm disease in goats may appear twelve to fifty-nine days after the introduction of the brood of *Str. capillaris*, permits the conclusion that sexual maturity may be attained in a shorter time than eight weeks. (*Str. micrurus*, according to Tapken, needs six to seven weeks for its complete development.) The location where the worms attain full sexual development varies more or less for different species. *Str.*

*filaria* is found preferably in bronchi of medium and small size; *Str. commutatus* is found in the smaller bronchi and penetrates into the pulmonary tissue after having deposited its eggs; the same is true of *Str. capillaris*, which is found in the small bronchi and alveoli. *Str. micrurus* is found preferably in the medium-sized bronchi (Jöst). The usual habitat of *Str. paradoxus* is in the medium and smaller bronchi; but occasionally it is found in the large bronchi and even in the trachea. Other lungworms inhabit the medium and small bronchi. *Trichosoma aerophilum* is a parasite of the trachea and of the large bronchi, while the sexually mature individuals of *Str. vasorum* are found in the right side of the heart and the pulmonary artery; those of *Ollulanus triocuspis* in the gastric mucosa.

The power of resistance of the worms appears to be very poor directly after the embryos are set free out of the egg shell; especially embryos of *Str. filaria* and of *Str. micrurus* perish within a short time under unfavorable conditions (putrefactive processes, desiccation), while otherwise they remain alive for weeks and go through one moulting in pure water. After the first moulting, the brood, probably now in a rhabditis-like intermediary form, attains great resisting power, so that it may live in water for months (Railliet, Baillet, Cohn), and may revive after a long period of desiccation. Larvæ of *Str. filaria* were found alive after sixty-three hours of desiccation (Railliet), after a year (Ercolani, Piana), and those of *Str. commutatus* after fourteen months. Sexually mature worms and their larvæ are not very resistant, if brought in contact with antiparasitic substances. Piana's investigations have shown that the larvæ are killed at once by an iodine-iodide of potash solution (1:10:100) and by a 4% corrosive sublimate solution; by vapors of iodine in one minute; by 15% solution of common salt in ten minutes; by a 10% solution of potassium iodide in fifteen minutes. Oil of turpentine, creosote and calomel apparently do not influence the larvæ, which, however, do not revive after desiccation followed by soaking in water. Absolute alcohol immobilized them for awhile, but they became motile again after some time.

The **natural infection** usually takes place with feed and water in pastures which have been contaminated by sick animals and which are sufficiently moist to favor the development of brood. The biologic properties of lungworms bring it about that pastures, once contaminated, remain dangerous for a long time, because the subsequently affected animals keep on furnishing the infectious material. Hence the disease becomes stationary in many parts. Other parts which are free from epizootics of domestic animals may become infected by wild animals.

Primarily dangerous are low, marshy pastures and those exposed to repeated inundations, while pastures which are situated favorably are dangerous only during permanent wet weather, even after contamination has occurred. The disease occurs under the latter conditions only in occasional outbreaks and at prolonged intervals which may extend to several years. Long lasting rainy weather naturally increases the danger, even in lowlands, and during very wet years the disease sometimes assumes an epizootic character over a larger territory. The disease is observed exceptionally in dry years. Under such circumstances the infection may occur by the inhalation of worm larvæ which may get into the air with dried and pulverized swamp material, or with material which by rain and wind may have been deposited previously on feed plants.

Infection in the barn is also possible. A porous and continually moist soil of improperly arranged hog pens, also the soil of the stables of other animals, the moist soil in the neighborhood of drinking places, stagnant water in the latter, offer a chance for the further development of embryos which are coughed up by infected animals. Indeed, lungworm disease has repeatedly been observed in young pigs which have never left the pen (Schultz, Moussu, Marek), or in cattle kept permanently in the barn (Kasperek, Scheibel). Sucking animals may occasionally infect themselves from the udders of their mothers which have become infected through contaminated straw. Exceptionally an infection may occur through dry feed contaminated with desiccated but living larvæ.

The brood of the worms is usually taken up into the body of the host in spring, at the beginning of pasturing; newer observations, however, permit the conclusion that infection may also occur in summer and even late in the fall (Schultz). According to Docter, field hares infect themselves in fall. Repeated invasions are not at all rare.

Direct contagion does not occur; experiments to infect healthy animals through the introduction of bronchial mucus containing embryos into the respiratory passages or into the stomach, or by the intravenous introduction of embryos and ova (Leuckart, Schlegel), were not successful.

Susceptibility. Young animals who have at least reached an age of several weeks are preferably affected. The observation of Kasperek, according to which calves one and one-half to eight days old were affected, has remained unique. Older animals are affected more rarely in direct proportion to advancing age. Sometimes, however, sheep and goats are affected equally and independently of age, especially in infection with *Str. capillaris*. The clinical symptoms are, however, usually much milder in older animals.

**Pathogenesis.** With the ingestion of food and drink the larvæ get into the stomach, and from there, during rumination or by active wandering toward the pharynx, they get into the respiratory passages. Zürn, Spinola and Csokor claim that the worm brood may also get directly into the air passages by the inhalation of dust from contaminated pools and marshes. The idea that the larvæ may get directly into the lungs with the blood current has been generally abandoned. Joest, however, believes that this mode of invasion, with a subsequent migration of the brood from the lung into the bronchi, cannot be entirely denied. *Strongylus* larvæ which get into the bronchi, probably invade then the smaller bronchi, some also the pulmonary alveoli, and produce, partly by their motion, partly by their metabolic products, an inflammation of the invaded bronchi (bronchitis verminosa) or of the alveoli. Since the invasion is usually not very intense, and since the degree of inflammation and the num-



ber and size of the inflammatory foci stand in direct relation to the amount of the worm brood, either disseminated and generally small inflammatory foci are developed in the lungs (bronchopneumonia verminosa lobularis) or the inflammation remains confined solely to the infected bronchus. During their further development, most lungworms (excepting *strongylus capillaris* and *strongylus commutatus*) try to enter the larger bronchi, where they reach sexual maturity and also cause an inflammation which, however, is mostly due to the deposited ova or the escaped embryos. Embryos may be aspirated into the smaller branches of the larger bronchus or wander there actively and after perforating the wall of the bronchioles, get into the lung parenchyma, where they produce inflammatory changes, and in this manner inflammatory foci develop in large numbers and in considerable extent (bronchopneumonia lobularis disseminata). However, the spread of worm pneumonia probably depends partly upon the auxiliary action of bacteria (Joest). In consequence of perforation of the bronchial wall by the embryos, air may get from the bronchial lumen into the lung parenchyma. But it is questionable whether interstitial emphysema of the lungs, which is seen frequently in cattle in connection with lungworm disease, is mainly due to perforation of the bronchial wall, as claimed by Joest, since the bronchial lumen is usually obstructed by masses of exudate before perforation occurs. Paroxysms of cough due to the existing bronchial catarrh appear much more important. Sexually mature worms (*Str. capillaris* and *Str. commutatus*) invading the lung tissue or embryos (*Str. vasorum* and *Ollulanus tricuspis*) by way of the blood current give rise to the formation of miliary or somewhat larger inflammatory foci.

From the bronchi the inflammatory process usually spreads to the peribronchial tissue and even further, and the diminution of elasticity of the bronchial walls induced in this manner, gives rise to a dilatation of the larger bronchi. Occasionally some bronchi are obstructed by masses of worms and secretions, causing atelectasis of the affected portion of the lung, which again forms the basis for a bronchopneumonic focus. If embryos are swallowed in great numbers, they may interfere with digestion.

From the investigations of Lignière one may conclude that lungworm disease furnishes a favorable soil for the development of secondary infections, particularly to the bipolar bacilli of hemorrhagic septicemia.

Extensive bronchial catarrh or bronchopneumonia cause disturbances of nutrition in consequence of an increased decomposition.

**Anatomical Changes.** These are quite variable according to the number of lung worms, to single or repeated invasions, to



the species of the parasites, the duration of the process, etc. As a rule, airless foci are found in the otherwise normal or emphysematous and pale lung tissue, preferably in the anterior and inferior portions of the lungs, from the size of a pea to that of a walnut. These foci are generally wedge-shaped, with the base on the pleural surface and the apex directed toward a larger bronchus. The foci are either sunken in and like meat (atelectasis) or more or less prominent, doughy, soft to tough, according to the duration of the process. They are reddish or grayish red, or even grayish yellow in color (worm-nodules, bronchopneumonia verminosa lobularis). On section these foci discharge a thick, reddish fluid which contains epithelia, pus corpuscles, ova, embryos and fragments of adult worms.

Not uncommonly extensive inflammatory areas are encountered, especially in certain species of animals (goats, sheep, calves and field hares), which are confined either to one or exceptionally to several lobes (bronchopneumonia verminosa lobaris sive diffusa). The affected portions of lung are like the spleen in color and consistency and they show on section dark red to black-red hemorrhagic spots, also dirty gray, crowded, even confluent spots. The expressed juice contains a large number of ova and worm embryos. In acute cases in calves the exudate often has a fibrinous character. In certain cases, the lungs contain vesicular nodules (as a rule, situated under the pleura) from the size of a millet seed to that of a hemp seed, which are found toward the apices and margins and which are of tough consistency (bronchopneumonia nodularis pseudotuberculosa s. alveolitis nodularis verminosa). These nodules are formed around an isolated, fully developed strongylus capillaris or a strongylus commutatus, or around aspirated ova. The nodules are light yellow to gray yellow in the presence of *Str. capillaris*, and red-brown to violet or blackish in the presence of *Str. commutatus*. After the death of the worms, the nodules undergo caseation and finally calcification.

In dogs pinhead-sized, translucent, pearl-like, exceptionally larger, bronchopneumonic foci are formed at the root of the lungs, sometimes also in other places; they are produced by the ova or embryos of *Str. yasorum*. In the presence of *Str. pusillus* the lungs of the cat show a similar picture; the embolic transportation of embryos of *Ollulanus tricuspis* produces nodules similar to miliary tubercles with a hepatized pulmonary parenchyma in their neighborhood.

The bronchi contain an abundant mucoid or mucopurulent secretion, sometimes streaked with blood, in which the sexually mature worms are found. Under the microscope numerous ova and embryos are seen. One frequently sees the first worms at the end of the trachea, i. e., at its bifurcation, and these may completely obstruct the larger or the medium-sized bronchi, in dense masses, which sometimes contain only a few or no worms at all. The mucosa is swollen, reddened and sometimes streaked with hemorrhages. Bronchiectases of varying sizes are rarely missed in the presence of worms.

Inflammation of the deeper layers of the bronchial wall can be recognized by its thickness, sometimes also by the presence of small purulent foci (peribronchitis). In such cases one finds a proliferation of pulmonary tissue around the bronchi, and white cicatricial bands are seen radiating from the bronchial wall into the air-containing tissue.

In hogs particularly pathologic changes are sometimes found only in the bronchi. These, however, frequently lead to atelectasis of the neighboring pulmonary parenchyma.

According to the observations of Osler, Blumberg and Rabe, *Strongylus canis bronchialis* produce in dogs wart-like nodules up to the size of a bean; they are arranged like mushrooms on the surface of the trachea and of the larger bronchi (tracheobronchitis verrucosa verminosa); these nodules contain worms which may project from under an uneven surface. In the pulmonary parenchyma there are many gray subpleural nodules, like granules of sand, and these contain each one worm. Osler also observed extensive bronchopneumonic inflammatory foci.

The pleura pulmonalis of the affected animals frequently appears mottled, thickened, but rarely covered with fibrinous deposits, or adherent to the pleura costalis. According to Kitt, the *Str. commutatus* sometimes produces pleuritic abscesses.

In severe cases the other organs present evidences of anemia and cachexia, especially subcutaneous edema and exudates into the serous cavities. A very intense gastritis is, according to Schlegel, of frequent occurrence. The bronchial glands are usually swollen.

**Symptoms.** The appearance of lungworm disease is possible at any time. It occurs, however, preferably in spring and fall or during the period of pasturing. The climax is generally reached in fall.

The reports of various authors on the appearance and occurrence of lungworm disease do not quite agree with each other. According to Railliet it is observed mostly during the warm season; according to Neumann, it occurs all the year around, especially between March and October; according to Zürn, in spring and in fall; according to Friedberger & Fröhner generally in fall; according to Dieckerhoff in calves toward the end of the pasturing season or even after its termination. Csokor states that lungworm disease in lambs appears in summer; in older animals only in fall. Moussu claims that the disease is prevalent all the year around, but reaches its climax late in the fall.

The period between the ingestion of the worm brood and the outbreak of the disease varies considerably according to the intensity of the invasion and to the individual power of resistance of the infected animal. Six to eight weeks elapse in the majority of cases till the symptoms become manifest. After a minor invasion and in strong adult animals the symptoms of disease come on after several months, while after a severe invasion, symptoms may be noticeable after some days. Schlegel saw the disease 12 to 59 days after the infection of goats, while Kasperek saw, as already mentioned, bronchopneumonic foci in infected calves only  $1\frac{1}{2}$  to 8 days old.

The disease manifests itself, as a rule, in all species of



susceptible animals as an insidious bronchitis, which increases in intensity from week to week, and which, in a portion of the cases, is accompanied by the symptoms of catarrhal pneumonia which finally leads to cachexia.

In **sheep** and **goats** one first observes cough which in the beginning occurs only in few animals, after long intervals and preferably during exercise. The cough is short, dry and strong and spreads gradually to the greater portion of the herd. At the same time it becomes more frequent, weaker and tormenting. In severe cases one observes real paroxysms of cough. The bronchial secretion is expelled in large lumps through the mouth and in the former are found the worms, their ova and embryos; the latter by microscopic examination. The nasal secretion is usually seromucoid, but it does not usually contain embryos or ova. Sheep frequently rub their noses on the ground; sometimes with such intensity that a loss of substance occurs.

The respiration becomes more and more difficult, especially in sheep, so that the animals finally breathe convulsively. A variety of râles are heard over the thorax and trachea. They are sometimes so loud that they are audible at a distance of several steps. Percussion usually shows nothing abnormal; but on percussing very attentively one occasionally finds dullness in circumscribed or even in more extensive places of the thorax, and sometimes also tympanic sounds. Bronchial breathing may likewise be heard. Ben Danon observed albuminuria. In extensive involvement of the lungs the temperature is elevated up to 40.5° to 41° C.

Emaciation and pallor of the mucosæ occurs in the further course of the disease, also a diminution in the elasticity of the skin. The disturbances in nutrition are often intensified by a complicating diarrhea. The embryos of *Str. capillaris* are always found in the feces (Schlegel). Finally edema appears on the entrance to the larynx, on the eyelids, the lips or on the whole front of the head; also on the lower thorax and the extremities. The animals become very weak and often fall down; when attempting to get up, the hind legs appear paralyzed and the patients succumb in complete prostration.

In **cattle**, especially in calves, one usually observes cough, at first strong and occurring at long intervals, but in the further course it becomes more frequent and more forced; when coughing the animals protrude the tongue from the mouth and expel masses of mucus which is sometimes mixed with blood and always contains worms. In severe cases violent paroxysms with attacks of suffocation occur several times a day and one of them may lead to death by asphyxiation. The respiration is more or less accelerated from the start, later on forced. There may, however, be rapid variations in the respiratory disturbances, especially during the hot season (Hartenstein). Percussion and auscultation show conditions on the whole similar to



those observed in sheep. A febrile temperature exists only exceptionally (Scheibel).

After an intense invasion the symptoms may increase in severity so rapidly that the animals succumb within three to eight days. The clinical picture, however, develops slowly, as a rule; the cough becomes gradually weaker, the respiration more and more accelerated, the ingestion of food less. Emaciation, anemia and edematous swellings make their appearance.

In **hogs**, lungworm disease occasionally does not lead to disturbances of health. However, *Str. paradoxus* sometimes causes great losses, probably after an intense invasion, and the epizootic disease then takes a course similar to that in sheep (Sequenz, Czokor). In other cases the affected animals show only disturbances of nutrition.

In **horses** and **asses** the disease occurs exceptionally and only leads to a clinical picture, similar to that seen in verminous bronchitis of calves. Fatal cases have been observed repeatedly in asses (Stewart).

Repeated attacks of respiratory difficulties are observed in **dogs** infected with *Str. vasorum*; these disturbances may disappear within a number of days or they may lead to the death of the animal. Ascites is developed in some cases. The disease occurs rarely, and generally only sporadically; however, an enzootic prevalence with numerous fatal cases has likewise been observed. Rabe observed obstinate cough and marked dyspnea in the presence of *Str. canis bronchialis* in the trachea and bronchi; Osler's Montreal cases occurred almost exclusively in young dogs and were characterized by fever, lack of appetite, weakness, paralysis of the hind extremities, convulsions, dry, short cough and vomiting. Railliet believes, however, that the bronchopneumonia found in these cases on post-mortem examination was due to distemper.

*Spiroptera sanguinolenta*, which occasionally may get into the respiratory passages, also produces chronic catarrh.

Lungworm disease of **cats** produced by the ova and embryos of *Str. pusillus* leads to frequent cough, often accompanied by vomiting, emaciation, diarrhea, cachexia and a fatal issue after two to three months. Catarrhal pneumonia may occasionally be produced by the embryos of *Ollulanus tricuspis*. Müller & Neumann found *Trichosoma aerophilum* in the presence of catarrhal changes in the lungs of cats.

Lungworm disease is very rare in **rabbits** and in these animals leads to the same symptoms as in wild hares, where it frequently prevails to an epizootic extent, viz., accelerated and difficult respiration with frequent and dry cough and gradually increasing emaciation. The disease often ends fatally.

**Course.** The course varies very much, according to the intensity of the invasion and the individual species of animals. The lungworms may frequently live in large numbers in hogs, but also not uncommonly in other animals, without disturbing the condition of health of the infected animals. Sheep and goats generally are affected most seriously after a more intense invasion, while the symptoms are milder in cattle and particularly in hogs. Among infected cattle, only weak calves



will succumb, while hogs die from the disease only in very exceptional cases. The course of the affection is influenced by the occurrence of secondary infections.

The duration of the disease also varies a good deal; in the great majority of grave, and hence unfavorable, cases, the duration may be two, three or four months. Deviations in either direction are, however, frequent. On the other hand, the disease may last more than four months, even over one year, particularly in adult strong animals; while on the other hand, it may take a fatal issue within a few days or weeks (without exception only in younger animals, particularly in calves). In such cases one generally finds a larger bronchus obstructed by masses of worms, or a rapidly spreading bronchopneumonia. In its mild form the disease ends in recovery. In cases of medium intensity recovery occurs, usually in hogs, frequently in calves, after regulation of the diet; while sheep and goats, everything else being equal, generally succumb, especially younger, weakened animals, or those which are in the later stages of pregnancy. The very severe forms usually end fatally. The advent of improvement is marked by a gradual diminution of the cough and by amelioration of the other symptoms.

Recovery is not always complete. Some animals, especially sheep and goats, still suffer in their nutrition after the disappearance of the catarrhal symptoms, and there may be a chronic wasting away with final death, unless the animal is slaughtered in time.

**Diagnosis.** Neither the symptoms of bronchial catarrh nor the subsequent cachexia are to be looked upon as characteristic symptoms of the disease. A diagnosis can only be made after the detection of the worms or their ova or embryos. The worms can be recognized with the naked eye in the coughed up sputum; if they are absent microscopic examination will detect the ova or embryos if present. Embryos of lungworms are also found in the feces of infected animals (Schlegel, Piana, Eichhorn).

Since cattle often swallow sputum which has been coughed up, Andersen recommends, after an attack of cough, the introduction into the pharynx of a rod armed at one end with a cotton plug; the secretion obtained on the plug of cotton is then examined. Bergeon has used this method with advantage. Hasenkamp's lung-mucus catcher might also be used with advantage. In an emergency masses of secretion may be removed from the pharynx with the hand.

Symptoms which are more or less similar to those of lungworm disease are found in catarrhal pneumonia from other causes, as well as in enzootic pneumonia of young animals. These affections, however, lead from the start to more or less marked disturbances of the general health with febrile elevation of temperature and they do not lead to the formation of abundant masses of secretion; both forms of pneumonia usually occur in very young animals. Differential diagnosis between cases of lungworm disease associated with bronchopneumonia

and other forms of pneumonia is only possible upon the detection of the worms and their broods. The disease is distinguished from pleuropneumonia by its usually afebrile course, by the absence of extensive areas of dullness and of pleurisy; further by the fact that cough remains for a longer time strong and convulsive. In young animals affected with pulmonary tuberculosis, the cough soon becomes weak. In *Estrus* disease symptoms of involvement of the lungs are absent.

**Prognosis.** This depends upon the severity of the symptoms, also upon the age, nutrition and species of the affected animal. Everything else being equal, the prognosis is most unfavorable in the case of sheep and goats, most favorable in lungworm disease of hogs. Fatal cases are always more numerous among young than among adult animals. Sometimes, however, there may be no difference in this respect (especially in infection with *Str. capillaris* or *Str. commutatus*). In lungworm disease of sheep the mortality varies from 10% to 70%.

**Treatment.** The most serviceable method of removing the parasites consists in intratracheal injections of antiparasitic drugs or in treatment with a spray apparatus also supplied with antiparasitic drugs. Favorable results have been obtained with these means in some cases. Some authors, however (Dieckerhoff, Tapken), deny that good results can be obtained with these methods. Oil of turpentine is adapted for intratracheal application, also tar preparations alone or in combination with the former. The following mixture may be used: olei therebent. rectif., ol. olivar. (ol. lini, ol. rapae) aa 100.0, creolin, purissimi 10.0. Of this mixture sheep receive an injection of 5.0 cc.; calves 15 to 20 cc.; the injection is repeated twice. Vaeth uses the following mixture with good results: Ol. caryophyll., ol. therebenthin., aa 100, acid. carb., ol. cadini aa 2.0 (calves 10.0 gm.). Wessel and Vaeth obtained favorable results in cattle, Krönig in lambs with injections of 20.0 or 50.0 of a 1% solution of carbolic acid; Bergeon in calves with creosote (creosote 20 parts, oleum amygdal. 100 parts, of this mixture 5.0; after four days, 20.0 for several days). Scheibel likewise cured cattle with creosote (creosote 1 part, spiritus rectif. and water aa 50.0). Nielsen used intratracheal injections of 0.1% solution of potassium picronitricum with good results in calves (according to the age of the animals from 20-60 in one dose).

Scheibel uses in the treatment of cattle in place of an injection syringe, a spraying apparatus which consists of an elliptically bent tracheal tube 0.4 cm. wide, with a shield and two eyes, so that the apparatus may be fastened with strings to the neck. The tube is armed with a trochar, so that it can be pushed into the trachea of the animal. The trochar is withdrawn and a dichotomously divided canula is then inserted into the tube. Creosote solution is then sprayed through the canula into the trachea and bronchi, just as is done when a Frick spray apparatus is used. The vessel containing the solution must be held lower than the canula, otherwise the fluid would simply run in without being finely divided. (Zwaenopoeel



and Coppens). The spray must be interrupted if an attack of cough sets in. The tube should be left in the trachea during the whole period in which this treatment is employed.

Full success can only be secured when the lungs are not seriously affected. Opinions are still divided as to the special form in which the drugs are to be used. Some observers prefer oil emulsions, since these remain longer in the air passages than the watery solutions. Nielsen, however, considers oil emulsions improper, since they do not mix with the mucus and do not get to the worms. The treatment with the spray apparatus as introduced by Scheibel appears most serviceable, since the finely divided fluid is, during inspiration, aspirated into all bronchi into which an air current is still entering.

Whether irritating smoke or vapors of similar action are indicated even in the beginning of the affection is very questionable. This method consists in burning masses of horn, tar, stinking oil, etc., on heated iron plates in closed spaces, where the animals are kept, or in heating turpentine until vapors are developed, and in compelling the confined animals to inhale the irritating air so as to produce much cough.

Stimulation and strengthening of the animals is of the utmost importance because experience has shown that strong animals sometimes survive even a severe attack. The animals should therefore have nutritious food, if possible cereals to which have been added bitters and preparations of iron. Pasturing animals should also receive nutritious food and hay. In order to prevent repeated invasions the pasture might be changed or the sick animals might be stabled. Animals which are very sick and which do not improve in spite of proper treatment should be slaughtered.

**Prophylaxis.** Lowly situated, marshy and damp pastures should be avoided. Care is particularly necessary in continued rainy weather, and young animals which are especially susceptible must be protected. In such neighborhoods where the disease has become permanently endemic, dry feeding offers the best protection (Schlegel). Preservatives which are praised as effective are useless, but one might water the animals before they are driven to the pastures, because this might cause them to avoid drinking water from pools and marshes. The sputum coughed up by the sick animals, the feces and the bedding-straw, the respiratory tract of slaughtered animals or those dying spontaneously must be destroyed. In a herd which is already infected, the young animals should be separated from the adults. Where hogs and cattle are concerned the stables and the drinking places should be disinfected. In places where the disease prevails among rabbits the weak and emaciated animals should be shot (Docter).

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## 5. Animal Parasites in the Air Passages of Fowl.

### (a) *Syngamus*. (Gapes.)

**Historical.** A disease caused in chickens and turkeys by *Syngamus trachealis* was first observed in 1779 by Wiesenthal in Baltimore. It has since been reported repeatedly and has been studied more carefully by a number of observers (Leuckart, Ehlers, Railliet, Mégnin, Walker).

**Occurrence.** The disease which is produced by syngami appears to be especially prevalent in America, England, Italy, France and Germany, and it prevails preferably in enzootic or epizootic distribution among pheasants bred in larger numbers in captivity. *Syngamus trachealis* invades especially pheasants, chickens, turkeys and peacocks, while *Syngamus bronchialis* infects water fowl (geese, ducks); however, *S. trachealis* occasionally affects other domestic birds, especially pigeons (Tossi) and room birds kept in cages. Syngami have also been found in partridges, American blue-jays, in cardinals, magpies, rooks, swallows, wood-peckers, storks, starlings and ravens. Young birds are generally affected.

Wiesenthal estimated that the loss by syngami in the invaded parts of the United States amounts to 80% of all young chickens; Crisp states that England loses annually about one-half million of chicks. In a large pheasant breeding establishment of France there was a daily loss of 1,200 pheasants. Klee estimates the loss in Germany to several hundred pheasants annually.

**Etiology.** Of the genus *Syngamus*, belonging to the family Strongylidæ, the air passages of birds are invaded by *Syngamus trachealis* Siebold (*Strongylus trachealis*, *S. primitivus*) and *Syngamus bronchialis* Mühlig. The air passages of water fowl sometimes contain *Monostoma flavum*.



Fig. 8. Ova of *Syngamus trachealis*.  
(According to Mégnin.)

*Syngamus* is a slender, red filiform worm. The head contains a mouth-opening surrounded by a strong chitinous ring. The posterior end of the much smaller male terminates in a ribbed bursa which covers two spicula. The female is three or four times as long as the male; its posterior end is blunt, the genital pore is situated in the anterior portion of the body. The male of *syngamus trachealis* is 2-6, the female 5-20 mm. long; the male of *syngamus bronchialis* 10 mm., the female 25 mm. The oval ova (Fig. 8) are small, provided with a double shell. The latter contains at either end a roundish opening closed by a delicate membrane. They contain a segmented mass or a developed embryo. In the trachea of fowl the male and female are usually found in copulation; the two look like one worm, branched under an acute angle.

**Infection.** Since the genital pore is closed by the adherent male, the ova or the embryos develop in the body of the mother,



and can get out only after the latter has become torn or decomposed, either in the air passages of the host or after having been coughed up into the outside world. In spite of the continuous copulation, the discharge of the ova is possible, according to Railliet. In moist material, in water or in the air passages eel-shaped embryos are developed in from one to several weeks. If ova or embryos are swallowed by fowl with food or drink, they adhere, according to Walker, to the wall of the esophagus or crop or they wander from there into the stomach, to get into the lungs and from there into the trachea, after perforating the wall of the esophagus.

Since it has been possible to produce the disease artificially (Ehlers) by feeding embryos containing ova, it must be assumed that the worm does not require an intermediary host for its full development, and that hence ova liberated in the air passages may there go through all developmental stages. It is therefore possible that the disease may be transferred directly from sick to healthy birds, if the latter swallow the infected secretion which the former have coughed up. Walker found embryos of *Syngamus* in infected neighborhoods in angleworms, which undoubtedly ingested them with soil; hence fowl may also infect themselves by the ingestion of angleworms. Klee has shown that rooks which frequently harbor the parasite may spread the disease, as may also magpies and starlings.

In a *syngamus-enzootic* among pigeons Rossi noticed a spread of the infection to the young squabs by the mother's feeding them. The squabs succumbed to a verminous inflammation of the crop.

**Anatomical Changes.** The worms are generally found in large numbers (30-40) in the air passages; *Syngamus trachealis* preferably in the trachea, *Syngamus bronchialis* deeper in the bronchi. The mucosa to which the worms adhere shows catarrhal changes, also occasionally small abscesses; the lungs are frequently affected in *Syngamus bronchialis* infections. Both species are not uncommonly found in the air vesicles.

**Symptoms.** The sick birds begin to cough and they shake their heads repeatedly; they expel masses of tenacious mucus from the mouth. They often open their bills (hence the common English name of the disease "gapes") and have a whistling breath. Emaciation soon develops in spite of a good appetite. The respiration becomes more and more difficult, abundant mucus collects in the buccal cavity, the appetite gradually disappears, the birds stand with ruffled feathers and they die totally exhausted or even earlier during an attack of dyspnea. Older birds sometimes recover if only few worms are present.

**Diagnosis.** The disease can be diagnosticated upon detection of the worms or ova coughed up with the secretion. The worms may be seen in the trachea of larger birds on intense illumina-

tion of the larynx, if this is pushed upward toward the buccal cavity with the trachea. In smaller birds they become visible, according to Renne, if after removal of the feathers, the trachea is drawn out with the skin covering it and held before a strong light.

**Treatment.** The worms which are situated in the upper portion of the trachea can sometimes be extracted by the aid of a long slender pair of forceps. Good results have recently been obtained by the intratracheal injection of a 5% solution of salicylate of sodium (Klee).

1.0 cc. of this solution is introduced with a syringe provided with an obtuse, bent needle. It is injected from the pharynx into the larynx or trachea. One may also push the needle into the trachea, which is held between two fingers. Since the trachea of smaller birds is hard to enter in this manner, it is sometimes necessary to lay it free through an incision into the overlying skin. The parasites drop off after the injection and are expelled by violent attacks of cough.

**Prophylaxis.** The expelled tracheal secretion, the feces and the cadavers of diseased birds must be destroyed; the floor and the poles of the coops must be disinfected; pools in the barnyard must be dried out; the feeding and drinking vessels must be cleaned repeatedly. Mégnin advises to spread denatured common salt on the floor (250 gm. to each 100 square meters of floor space). Sprinkling the soil with burnt lime may likewise be serviceable. Sick birds must be separated from healthy ones.

**Literature.** Klee, D. t. W., 1899, 465 (Lit.).—Neumann, Mal. paras., 1892, 587.—Railliet Zool. méd., 1895, 453.

*Syngamus laryngeus* occurs in the larynx of cattle in the southern provinces of Annam, but it does not produce any disturbances.

#### (b) The Air-sac Mite of Chickens. *Cytolichus Sarcopoides*.

*Cytolichus sarcopoides* (*Cytoides nudus*), a mite, 1½ mm. long, the female of which is oviparous, occurs occasionally in large numbers in all parts of the air passages, especially in the large air cells. The evolution of this mite is unknown. It occurs preferably in the air passages and larger air cells of chickens and pheasants, where it forms very small white, occasionally motile, points visible to the naked eye, which form a hoar-like deposit when much crowded together. Exceptionally the mites are also found in the air spaces of the bones; and the mucosa of the bronchi rarely becomes catarrhal under their influence. Then the mites are found in an abundant mucopurulent mass or in croupous membranes.

*Cytolichus sarcopoides* frequently does not produce any symptoms of disease, even if present in large numbers. In other cases it causes a catarrhal condition of the air passages with cough which, according to Zürn, is accompanied by a peculiar sound, as if a small foreign body had gotten into the trachea. In an exceptionally extensive

invasion there may be severe bronchitis with the formation of so much secretion that death may occur from suffocation (Méglin). Rosencrantz (B. t. W., 1909, 757) saw numerous fatal cases exclusively among cachectic adult chickens in whose peritoneal cavity there were innumerable sarcopteslike mites; inflammatory changes were however absent.

The disease cannot be diagnosticated from its clinical symptoms as long as the birds are alive. An effective treatment is unknown. Inhalations of tar or iodine vapors might sometimes be tried.

**Symplektoptes cysticola.** Kasperek saw an enzootic among pigeons caused by *Symplektoptes cysticola*. In distinction to previous references, according to which *symplektoptes* lives only in the subcutaneous tissue, it caused in Kasperek's cases an inflammation of the air passages, progressive inflammation and finally death of the animals. The mite-like parasites were found in the air passages; also yellowish, whitish, elongated nodules in the lungs, each containing one parasite.

**Literature.** Kasperek, D. t. W., 1907, 623.

## SECTION IV.

### DISEASES OF THE LUNGS.

#### 1. Congestion of the Lungs and Edema of the Lungs. *Hyperaemia et Oedema pulmonum.*

Hyperemia of the lungs consists in an abnormally high amount of blood in the pulmonary capillaries, which may be due to an increased inflow of blood (*hyperaemia pulmonum activa*) or the condition may be brought about because, for some reason or other, the blood cannot flow without impediment from the lungs toward the left side of the heart (*hyperaemia pulmonum passiva*). Edema of the lungs occurs in consequence of an extravasation of serous fluid into the alveoli and bronchioles and also into the interalveolar tissue.

**Etiology.** Hard labor may produce an increased supply of blood to the lungs, since the heart which works energetically and rapidly may bring an increased amount of blood into the pulmonary artery. Active hyperemia on this basis, therefore, frequently occurs in racers, in hunters and army horses, and in hunting dogs, especially in summer when the warm air also has a weakening effect upon the pulmonary vessels. In a considerable proportion of such cases we are, however, rather dealing with heat-stroke or with acute cardiac weakness due to passive hyperemia (for details about the disease called "summer foundering" by Bongartz, see in the chapter on heat-stroke).

The disease under discussion appears in horses and, perhaps, also in other animals in combination with rheumatic affection of the intercostal muscles, during long railroad transportation, particularly if the animals are crowded in the car (Dieckerhoff, Sigl). Overheating of the body, exertion and excitement are probably the injurious factors (Dieckerhoff describes this condition as a disease *sui generis* under the name of *Pleurodynia*).

Very hot air or air mixed with acrid and irritating vapors may produce an increased blood supply by dilating the pulmonary vessels or by acting as an inflammatory stimulus; this has been shown by observations made after conflagrations.



**Hyperemia** seen in the early stages of acute inflammatory processes also belongs to this group.

A collateral hyperemia is produced when the blood current **in the lungs** meets with a rapidly forming extensive impediment such as occurs in pneumothorax on the healthy side and in considerable elevation of intra-abdominal pressure on both sides.

Congestive hyperemia is one of the usual concomitant conditions in stenosis of the left venous ostium or in mitral insufficiency. In the majority of cases, however, it develops in consequence of cardiac weakness in dilatation of the heart, in non-compensated valvular disease of the heart in consequence of myocardial degeneration in many acute infectious diseases. Compression of the pulmonary veins in pericarditis, decrease of the negative intrathoracic pressure in bloating of the stomach or of the intestines may be the cause of congestion of the lungs. The prolonged recumbent position on one side may also cause hyperemia in the lower portions of the lungs without the contribution of the above factors (hyperaemia hypostatica), because in such a position the venous blood has to overcome the effect of gravity.

Basch and others have shown that hyperemia of the lungs with increase of the pressure in the capillaries makes the latter stiffer and so impedes the expansions of the pulmonary tissue; it is also claimed that the capillaries become stretched and thus bring about a certain amount of dilatation of the alveoli. The slackening of the blood-current in congestion also acts unfavorably.

Edema of the lungs is also due to various causes. All those factors which produce hyperemia of the lungs may also produce edema of the lungs if they are acting more intensely or over a long period of time. Those types of edema which are due to alterations of the vessel walls brought about by irritating substances are easily explained, for in these cases the extravasation of blood serum is a sequel of disease of the vessel walls as is generally the case in inflammations. A similar mode of origin of pulmonary edema is that seen in association with acute pneumonia and in the course of some infectious diseases (anthrax, malignant edema and other septicemic affections). Bacteria and their toxins then need not necessarily act directly upon the pulmonary tissue, but their inflammatory action may be exerted through the blood circulation. A similar effect may be manifested by certain vegetable or mineral poisons or by decomposition products of the organism; intense edema of the lungs which is sometimes seen in severe disease of the kidneys is probably of a similar origin.

In the majority of cases the edema is due to stasis of the blood. It frequently develops on this basis shortly before death from cardiac weakness, combined with insufficient expansions of the lungs. Valvular lesions in the left venous opening

often lead to edema of the lungs even when compensated, because the blood cannot flow freely from the veins of the right side of the heart even when the heart muscle acts well. Cardiac weakness exclusively of the left side is very rare as long as the right side contracts powerfully (Krehl). Hypostatic hyperemia may also lead to edema of the lungs.

Nutritive disturbances of the vessel walls in cachetic conditions likewise predispose to edema of the lungs.

**Anatomical Changes.** The consistency of the lungs is somewhat increased in pulmonary hyperemia and they are possibly also somewhat bloated, the cut surface is darker and discharges blood freely. If the congestion has lasted a long time the lungs become tough in consequence of an increase of connective tissue and the cut surface appears rust-brown owing to the presence of haematoidin granules (so-called *induratio brunea*). Hypostatic hyperemia is usually found only on one side or on part of one side; here the pulmonary parenchyma is of spleen-like consistency because the alveoli are filled with blood corpuscles and blood plasma. This condition is called splenization or, wrongly, *pneumonia hypostatica*.

Pulmonary edema is recognized by a fine foamy fluid, mixed more or less with blood, which oozes out in large amount from the cut surface of the lung which has become less elastic. The bronchi are filled by a similar fluid and the air contents is very much diminished. One also observes the usual signs of death from suffocation.

**Symptoms.** The external symptoms of pulmonary hyperemia and of edema are on the whole very similar and consist in the signs of rapidly increasing dyspnea and attacks of suffocation. The animals appear at first anxious and restless, but later on become listless and somnolent. The mucosae of the head become cyanotic, the eyes protrude and the jugular veins swell up. The respiration is much accelerated and forced and soon becomes rattling. In edema of the lungs the much dilated nostrils discharge a foamy, serous or serohemorrhagic fluid. A short dull cough is heard periodically. The percussion sound in hypostatic hyperemia is dull over the lower portions; otherwise it is normal or may be somewhat dull and higher in pitch on account of the increased tension of the intercostal spaces; in severe cases of pulmonary edema it is likewise dull, possibly here and there tympanitic. Vesicular breathing is lessened in hyperemia, or on the contrary, rough; in edema it is much weakened, sometimes entirely absent; crepitant and rattling râles may be heard. The heart beat becomes pounding, the accelerated pulse is at first tense and full, but later, or in concomitant heart disease from the start, small and weak.

All these symptoms may disappear as rapidly as they

came on, and the animals may completely recover within 6 to 12 hours, especially in active hyperemia, while in other cases suffocation comes on with the increase of the symptoms within an equally short space of time (apoplexia pulmonum). On the other hand congestive hyperemia usually develops gradually and, even in severe cases, leads to death only after several days. Pulmonary edema also causes, at times, death very rapidly and almost in an apoplectic manner (apoplexia pulmonum serosa).

In pleurodynia, respiratory disturbances, tenderness to pressure of the intercostal spaces and acute bloating of the lungs are observed, all symptoms being due to the tenderness of the intercostal spaces. These symptoms usually disappear in four to six days without leaving a trace, sometimes, however, a fibrinous or serofibrinous pleurisy develops subsequently, which may cause the death of the patient.

**Diagnosis.** Both hyperemia and edema of the lungs can usually be diagnosticated easily if one considers the usually sudden onset of the disease with the symptoms described and if a history or the signs of the external irritants mentioned or of an existing primary affection are obtained. In heat-stroke not only dyspnea is observed, but also marked cerebral depression, physical weakness and a considerable elevation of temperature. In septic infectious diseases (anthrax, swine erysipelas) the symptoms of a severe febrile general affection are present from the start, coming on without any discernible external influences. Edema of the lungs is distinguished from diffuse bronchitis by the absence of fever and by the presence of crepitant râles and a foamy discharge from the nostrils. In pulmonary hemorrhage foamy blood is discharged from the nostrils.

The **prognosis** is usually not unfavorable in active hyperemia occurring in otherwise healthy animals, if proper treatment is at once instituted. Passive congestive hyperemia, hypostatic hyperemia and edema of the lungs frequently terminate fatally. Symptoms of cardiac weakness are of unfavorable prognostic significance as is also elevation of temperature.

**Treatment.** Rest is most important for the patient. Extensive blood-letting considerably relieves the heart and diminishes the blood pressure in the lungs, so that they are again able to expand properly; blood-letting, carried out at the proper time, may save the life of an animal in acute pulmonary hyperemia; but even in congestion in the lesser circulation it is advisable to withdraw a moderate amount of blood.

Subcutaneous injections of excitants (camphor, caffeine, ether), rubbing or irrigation with cold water are especially indicated if the pulse is weak. Cardiacs are serviceable in



cardiac disease. As long as the pulse is sufficiently strong, one may try in edema of the lung subcutaneous injections of atropine (0.03-0.05 gm. for large, and 0.005-0.01 gm. for small animals), scopolamine (0.01-0.05 and 0.003-0.01 gm.).

By way of prevention, animals with cardiac disease should be protected against hard work, rapid running, etc.; their evacuations should be regulated. Sick animals which are down must be turned frequently.

## 2. Pulmonary Hemorrhage. Haemoptoe.

(*Blutsturz* [German].)

As pulmonary hemorrhages all those cases are designated clinically where the source of the blood is found in the air passages behind the larynx or in the lungs themselves; it is not possible to differentiate clinically hemorrhages arising from the bronchi or from the pulmonary parenchyma.

**Etiology.** Haemoptoe often is a consequence of hyperemia of the lungs (see page 84). Pulmonary hemorrhage is, therefore, seen preferably in horses after overheating in rapid running or in consequence of hard work, also as part of the clinical picture of heart disease. Guittard often saw pulmonary hemorrhage in young, well-nourished chickens after taking cold during rough weather.

Hemorrhages from the lungs, which occur frequently in the course of infectious diseases, can be explained by a diminished resistance of the vessel walls. Diseases of this kind are, particularly, purpura haemorrhagica, smallpox, anthrax, septicemia, pyroplasmiasis in horses, acute pneumonia, etc. Deficient nutrition of the vessel walls undoubtedly is a factor in such affections as leukemia and renal diseases and other diseases leading to cachexia. Frequent hemorrhages in hemophilia likewise depend upon a diminished tissue resistance.

Necrotic processes in the mucosa of the air passages and in the pulmonary parenchyma may produce hemorrhages by erosion of vessels. This occurs in glanders in horses and in tuberculosis in cattle, in pulmonary gangrene, in ulcerating neoplasms, and in cavity formation in the lungs which occurs sometimes after pneumonia (Fröhner, author's own observation).

Aneurysms of the aorta or of the pulmonary artery which happen to be adherent to a bronchus may rupture and produce a fatal hemorrhage. Thrombosis or embolism of the pulmonary vessels may likewise cause minor hemorrhages and this occurs often in valvular disease of the heart. A rare cause of haemoptoe are injuries to the lungs.



**Symptoms.** Hemorrhage of the lungs does not always become manifest because small amounts of blood may remain in the air passages and may be absorbed rapidly or they may coagulate and subsequently disintegrate. An insignificant hemorrhage manifests itself sometimes in such a manner that an existing nasal secretion or bronchial sputum contains small bloody dots or streaks, or that it looks saffron-yellow or rust-brown in consequence of an intimate admixture of blood. (A saffron-yellow color is seen particularly in pneumonia, a rust-brown color in valvular disease of the heart.)

Severe pulmonary hemorrhages lead sometimes to a frightful flow of blood out of the nostrils and mouth, and intense dyspnea is associated with such an occurrence. The flow is usually bright red and foamy and small air bubbles may even be seen in a coagulum which may have formed. The respiration is much accelerated and convulsively difficult; the animal betrays great anxiety, trembles, perspires, staggers and finally falls down if the hemorrhage continues. The mucosa, at first cyanotic, becomes gradually paler; the pulse becomes accelerated and filiform. Râles are occasionally heard over the lungs; also over the trachea; sometimes crepitant sounds are audible.

The significance of pulmonary hemorrhage depends primarily upon the amount of blood lost. Animals may look healthy after small amounts of blood are lost, larger animals even after the loss of three or four quarts; although they may show a transitory weakness. A considerable loss of blood leads more or less to the signs of grave anemia which disappears only after a long time or may lead to cachexia. Very excessive hemorrhage may, of course, terminate fatally in a very short time, within fifteen to thirty minutes.

**Diagnosis.** Hemorrhage of the lungs may be recognized from the bright red and foamy character of the blood voided. The source of the hemorrhage however is recognized only with difficulty. It may occur that blood coming from the posterior parts of the nose, as well as blood coming from the mouth, pharynx or esophagus flows backward to be then expelled with attacks of coughing. Under these circumstances the blood does not contain numerous air bubbles and the examination of the parts named and of the thorax will usually afford information about the source of the blood. In hematemesis the blood is expelled with vomitory movements, it appears dark in color, and is often mixed with coagula; it reacts acid and frequently contains particles of food. One must, however, not forget that the act of vomiting is brought about in smaller animals by coughing, that vomited blood may subsequently get into the air passages, that blood from the lungs likewise becomes dark later on and that a large amount of blood will remain alkaline even in the stomach. Hence, in making a diagnosis, it is neces-

sary to consider all circumstances and the condition of all internal organs.

**Treatment.** When a hemorrhage is not abundant, it suffices to take the animals to a moderately cool place, to protect them against every excitement and to give them complete rest; they should not be excited even by an examination. Hemorrhages occurring in young chickens may be stopped by bringing the animals to a warm place. If cough is present, it should be suppressed by narcotics, while the hemorrhage should be met by drugs which contract the vessels. Most commonly used are ergot (*secale cornutum*) (15.0-25.0 or 0.5-2.0; of the extract 5-10 or 0.2-1.0 gm. internally, the extract in half doses subcutaneously); ergotin (0.02-0.2 gm. hourly for dogs); *extractum hydrastis* (10-15 gm. for horses subcutaneously). Mellin and Plumier consider *secale cornutum* as not serviceable because it brings about contraction only in the larger circulation so that it may increase the pulmonary hemorrhage; fluid extract of ergot is said to be an exception from this rule. Water containing vinegar, turpentine or chloride of iron are serviceable for inhalation. Cold packs of the thorax and, according to others, warm packs may be employed. If the site of the hemorrhage can be made out, the affected half of the thorax may be placed at rest by applying strips of adhesive plaster to it. One may also employ Moerkeberg's rubber plates (6-8 mm. thick), which may be moulded easily after a previous soaking in hot water.

Venesection appears indicated only in the presence of hyperemia of the lungs; excitants and digitalis must be used when great weakness is to be counteracted. Subsequent anemia must be properly treated (see Vol. I). Animals which are predisposed to hemorrhage from the lungs must be protected prophylactically against excessive work, overheating, cold, etc.

### 3. Alveolar Bloating of the Lungs. *Emphysema pulmonum alveolare*.

The term emphysema of the lung designates an abnormal dilatation of the alveoli with an enlargement of the lungs due to it; the pulmonary parenchyma may either remain normal (*emphysema pulmonum alveolare acutum*) or atrophy may occur of the interalveolar septa (*emphysema pulmonum alveolare genuinum s. substantiale s. essentielle*).

#### (a) *Acute Alveolar Emphysema.*

##### *(Simple Bloating of the Lungs.)*

Acute alveolar bloating of the lungs consists in simple dilatation of the air vesicles without any structural changes of the pulmonary parenchyma. It is always secondary in nature



and may disappear completely as soon as the primary affection is relieved. It is therefore of minor clinical importance. According to the primary disease simple bloating of the lungs may be diffused over both lungs (*volumen auctum pulmonum*, Krehl) or it may be confined to portions of the lungs (*emphysema pulmonum alveolare vicarians*).

**Etiology.** Diffuse acute bloating of the lungs is a regular concomitant to diffuse microbronchitis. It is seen in the course of pleurodynia (see page 87); also in long-lasting convulsive cough, in long-continued strong inspiration and expiration as it is seen in certain diseases of the air passages or in a protracted agonal struggle.

**Circumscribed acute bloating of the lungs** develops if the bronchi of a certain territory become narrowed or if certain parts of the lungs have become obstructed. In the former case the corresponding portions become emphysematous; in the latter case, neighboring areas.

Diffuse, as well as circumscribed emphysema develops partly on account of overstretching of the alveoli, partly on account of the fact that the escape of air out of the alveoli is more or less interfered with in certain diseases of the air passages, hence more and more residual air remains in the alveoli.

**Symptoms.** Aside from the symptoms of the primary disease there is in diffuse acute bloating of the lungs, an increased resonant percussion sound over the lower and posterior pulmonary margins and a displacement backwards of the posterior inferior pulmonary border, which may be extensive enough so that the posterior inferior pulmonary border reaches to the costal arch (authors' own observation). The percussion sound is more rarely affected in circumscribed acute bloating of the lungs, i. e., when a larger lung territory has become affected. The respiratory sounds vary in both forms, according to the nature of the primary affection; if larger pulmonary territories have been obstructed one hears intensified vesicular breathing over the emphysematous portions.

Bloating of the lungs decreases the expansion of the lungs in direct proportion to its extent because severe bloating, which has existed for any length of time, will decrease the elasticity of the pulmonary tissue. Bloating, however, will disappear without leaving any trace, if the primary disease leads rapidly to recovery, but chronic alveolar emphysema with atrophy of the interalveolar septa will develop when the primary disease exists for a longer period of time.

**Diagnosis.** If the percussion sound is changed in the manner indicated above and if primary diseases as mentioned

are present a diagnosis can be made safely, provided chronic alveolar pulmonary emphysema can be excluded.

**Treatment.** This depends entirely upon the primary disease. Its removal must be sought for because its persistence entails danger of the development of lasting chronic dilatation of the alveoli.

(b) **Chronic Alveolar Bloating. Emphysema pulmonum alveolare genuinum.**

(*Heaves; Emphysema pulm. alv. substantiale.*)

Chronic alveolar bloating of the lungs consists in a permanent dilatation of the alveoli accompanied by atrophy of the interalveolar and the interinfundibular septa and of the vessels contained in them.

**Occurrence.** Chronic emphysema of the lungs is found preferably in horses, more rarely in working oxen, quite frequently in hunting dogs, usually in canines that are somewhat advanced in age. It is the most common cause of horses being broken-winded.

**Etiology.** Chronic emphysema of the lungs develops, as a rule, after the animals have been used a long time for heavy work (pulling or running); it is more rarely due to continued inspiratory or expiratory dyspnea in chronic diseases of the air passages. Since continued inspiratory dyspnea is rare as long as the air can stream into the lungs, it plays only an insignificant rôle in the production of chronic pulmonary emphysema. A more potent and more frequent cause of the affection is difficult expiration, which is occasionally simultaneous with inspiratory dyspnea.

Long-continued and convulsive cough is more dangerous in this respect than impeded expiration, because a deep inspiration precedes every effort at cough and this produces a high air pressure in the lungs. On account of narrowing of the bronchial lumen by plugs of mucus or swelling of the mucosa, the escape of air out of the alveoli is impeded in catarrh of the finer bronchi. These circumstances sufficiently explain why chronic bronchial catarrh so frequently leads to emphysema of the lungs. Ball saw several cases of chronic emphysema in cats following multiple bronchial adenomata.

The presence of chronic bronchial catarrh in emphysema of the lungs does not per se prove that the latter is always caused by the former, because emphysema may exist and only subsequently lead to bronchial catarrh.

Emphysema of the genuine or substantial type may arise also from other causes aside from those already enumerated, and a permanent dilatation of pulmonary alveoli with atrophy of the pulmonary tissue may



develop as a vicarious chronic emphysema of the lungs in various chronic diseases leading to the obstruction of portions of the lungs. This form is of clinical importance only in so far as it increases still further the respiratory difficulties caused by the primary disease.

**Predisposition.** The frequency of the disease increases with age. It is exceedingly rare in horses younger than five years, but common in older horses. The frequent occurrence of the disease in older animals finds its explanation in the fact that forced respiration continued over longer periods and due to hard work will produce a detrimental effect; work horses are moreover frequently exposed to affections of the respiratory organs and to disturbances of nutrition in consequence of insufficient feeding and of digestive anomalies.

The influence of insufficient nutrition is seen convincingly in senile atrophy of the lungs, where dilatation of the alveoli is due exclusively to atrophy and thinning out of the inter-alveolar septa. The power of resistance of the pulmonary tissue shows a good deal of individual variability because it would otherwise be unexplainable why some animals contract the disease after a comparatively short exposure to harmful influences and at a comparatively young age, while others remain well under the same conditions or only develop a mild form of the affection late in life. A diminished resistance of the pulmonary tissue may be acquired, congenital or hereditary.

**Pathogenesis.** A considerable dilatation of the alveoli occurring again and again innumerable times, perhaps for years, will decrease the elasticity of the pulmonary tissue of itself to a certain degree. Much more important, however, is the circumstance that under the conditions indicated the alveolar pressure becomes increased during expiration or coughing and the interalveolar and interinfundibular septa with their capillaries, are compressed and distorted from both sides. Frequently recurring narrowing of the pulmonary capillaries impedes the free flow of the blood more or less and some capillaries will become impervious, while blood corpuscles are arrested in them. Since there is then no blood-current in some capillaries or at least only a current of blood plasma, the nutrition of the pulmonary parenchyma suffers, including that of the compressed and distorted capillaries. For this reason and in consequence of the ever increasing alveolar pressure the capillaries become completely obliterated, the elastic fibers yield to the increased pressure and the alveolar epithelia undergo fatty degeneration. Thus the interalveolar and interinfundibular septa gradually become thinner, the interstices between the yielding elastic fibers become larger, until finally the interalveolar and later also the interinfundibular septa disappear entirely so that neighboring alveoli become confluent to form a common cavity.

If this process goes on in the lungs it leads to a successive decrease of elasticity, hence the lungs will increase their size with difficulty during respiration and also decrease it with difficulty in expiration, so that a gradual increase in the volume of the lungs is produced. The obliteration of a portion of the pulmonary capillaries which bring about the exchange of gases, leads to a decrease of the respiratory surface, to an increase in pressure in the pulmonary artery and consequently to a hypertrophy of the walls of the right side of the heart. The decrease of elasticity and the diminution of the respiratory surface cause dyspnea which is particularly manifest in expiration. Deficient ventilation of the air passages, and changes in the lesser blood circulation predispose the animals to bronchial catarrh.

**Anatomical Changes.** The lungs appear larger, softer and less elastic (more like an air-cushion) and in consequence of the diminished blood-supply also paler than normal; their surfaces frequently show the impressions of the ribs (this is important from a forensic standpoint). The changes are most marked toward the inferior and posterior borders. The diaphragm appears raised posteriorly, the heart is covered by the lungs to an increased extent. The margins of the lungs are rounded off, individual air vesicles can be recognized with the naked eye and between them sometimes larger air spaces, up to the size of a hazelnut. If cut into, the lung tissue collapses more slowly than under normal conditions and without crepitation; the cut surface is pale red and discharges only a small amount of foamy blood; small droplets of pus, which appear on pressure, point to the existence of bronchial catarrh. The enlarged lungs are lighter in weight than those of healthy animals.

Stömmer has investigated the histologic changes and has ascertained that the diameter of the alveoli is increased from a normal of 0.15 mm. to 1.5 mm., while the diameter of the interalveolar septa is decreased from  $8\ \mu$  to  $1.2\ \mu$ . The vessels are more straight, their lumen is narrowed, partially obliterated. The number of elastic fibers is decreased, the alveolar epithelia are in a state of fatty degeneration. Rindfleisch found in the lungs of man that new vessels are formed partially in place of those that have become obliterated; this change is brought about by a new formation from branches of the pulmonary artery which enter into anastomosis with branches from the bronchial artery. The involuntary muscle fibers of the bronchial wall are increased.

Hypertrophy of the right cardiac ventricle is found in advanced cases, in the most severe cases also dilatation with symptoms of general congestion.

In senile atrophy of the lungs (atrophia senilis pulm.) the picture is similar, because the alveoli have become enlarged secondarily, ex vacuo, in consequence of tissue atrophy. Such lungs are, however, never enlarged. They are, on the contrary, smaller and there is no hypertrophy of the right side of the heart.

**Symptoms.** With the usual methods no changes can be demonstrated in the lungs while the disease is in its stage



of development. On close inspection one may recognize a minor change in the respiratory movements; the expiration is somewhat prolonged and occurs with the auxiliary action of the abdominal muscles. In less well nourished animals one sees during inspiration a slight sinking in of the intercostal spaces and of the wall of the thorax in the cardiac region. The frequency of respiration is at first not changed. The animals are, however, more easily tired during work, and they breathe then more rapidly; they show in short the symptoms of being short of wind even at this early stage.

In a more advanced stage the dyspnea, however, becomes distinct. The ribs are now elevated strongly and moved forward, and there is strong passive sinking in of the intercostal spaces of the lower portions of the thoracic wall, also of the anterior aperture of the chest and of the flanks. Expiration occurs under very strong auxiliary action of the abdominal muscles and the formation of a depression along the costal arches. The expiration is often double and is accompanied by an upward movement of the lumbar portion of the spinal column and by a shaking of the whole rump. The inspiration often also occurs in two stages (see Fig. 10). The respiration is usually somewhat accelerated.

A lasting dilatation of the thorax progresses in like degree with the gradual dilatation of the lungs. The more prominently curved ribs produce a barrel-shaped thorax, a picture which betrays the character of the disease on first sight.

The percussion sound over the lungs is deeper and markedly loud. The increase in intensity is particularly marked over the inferior margins of the lungs, where a higher but duller sound is heard under normal conditions. The area of cardiac dullness becomes smaller or may disappear entirely. In severe cases one can also show by weak percussion that the posterior boundary of the lungs has been displaced backward and downward. (In healthy horses which are not overly fat one finds the posterior boundary of the lungs in the iliac line where it is crossed by the seventeenth rib, in the ischial line by the fifteenth rib; in the shoulder line by the eleventh rib; and it passes into the lower horizontal boundary in the seventh intercostal space; in emphysema of a high degree, however, the lungs reach to the eighteenth rib in the iliac line, to the seventeenth rib in the ischial line, and to the twelfth or fourteenth rib in the shoulder line. [See Fig. 9].)

On auscultation one hears weakened vesicular breathing, often mixed with dry or moist râles as an indication of an existing bronchial catarrh, which then also leads to an intensification of the vesicular breathing sounds over some portions of the thorax.

Cough is due to a more marked bronchial catarrh and usually occurs in a somewhat more advanced stage of the disease. It is weak in more pronounced cases of emphysema

on account of diminished pulmonary elasticity, short, and as a rule, dull.

The apex beat is frequently somewhat accelerated, the heart sounds are weakened, the second pulmonary sound is commonly somewhat accentuated (felt in the median portion of the left lower thorax in the third intercostal space). Transitory signs of cardiac weakness may come on, particularly after hard work.

The respiratory difficulties increase markedly during work and this reduces the work-value of the animals correspondingly with the advance of the disease, when the signs of shortness of breath become very marked.



Fig. 9. Displacement of the posterior, lower boundary of the lung in chronic emphysema. The anterior finer line indicates the normal boundary which at C goes over into cardiac dullness; the heavier line indicates the boundary of the enlarged lung. The figures indicate the numbers of the ribs.

The investigations of Richter and Schmidt have shown that shortness of breath produced by chronic alveolar pulmonary emphysema, particularly during exercise, brings about an elevation of temperature which will return to normal only after two hours. Half an hour after the exercise the temperature may still be 38.9° C.

Disturbances of nutrition and permanent circulatory disturbances appear after the disease has lasted a long time. Animals which were previously fairly well nourished or even fat gradually become emaciated; edema appears on the lower



abdomen, on the lower chest and on the extremities, until the animals are utterly unable to work and have to be killed.

**Course.** Months, perhaps years, elapse until the disturbances in respiration and circulation point, even at rest, to material tissue changes. (Holterbach saw severe shortness of breath appear forty days after a convulsive cough; it is not clear, however, whether this case was one of chronic emphysema of the lungs.) The course is much influenced by the work required of the animals, since hard work leads to respiratory and circulatory disturbances and to a more rapid progress of the pulmonary changes, to the development of complications, particularly of obstinate bronchial catarrh and these of course materially contribute to a further deterioration of the condition of the animals. This may particularly be observed in horses which, because of their lessened ability for work are sold to poorer proprietors, where they have to work still harder and at the same time receive poorer food, for which reasons they often succumb rapidly. Recovery or a permanent stationary condition of the affection appears excluded on account of its very nature, because the existing pulmonary bloating will produce respiratory difficulties and these will of necessity further increase the morbid condition. Transitory, and even marked and rapid improvement occurs, but this is almost without exception due to improvement in the complicating bronchial catarrh. If this occurs, a decrease of the pulmonary dilatation is at once demonstrable. A sudden deterioration may occur in consequence of the development of an interstitial emphysema.

**Diagnosis.** In a more advanced stage the disease can easily be diagnosticated, if the signs of enlargement of the lungs, of an increased air content, and a decrease of elasticity are present. Early in the course of the disease it cannot be diagnosticated by the aid of physical methods, because the changes in the pulmonary tissue are of a minor degree; it may, however, be suspected if dyspnea of a more or less pronounced character is present and if reliable data reveal a preceding pulmonary affection, while careful examination of the thoracic organs for other changes is negative.

Temporary relief from dyspnea is often brought about by horse dealers by the internal administration of leaves, roots or seeds of plants containing atropine (*Atropa belladonna*, *Datura stramonium*, *Hyoscyamus niger*). Raitsits' experiments made in the Budapest clinic have shown that these plants do not merely reduce the frequency of respiration, but also abolish the double period and the forced character of the respirations, so that it may appear perfectly normal, even in advanced cases. The effect is produced within fifteen minutes and lasts a whole day. After the effect has vanished the dyspnea becomes more intense than previously. During the first hours after the administration of the above-mentioned plants the buccal mucosa is very dry (to mask this it is usually rubbed with fat). One also observes dilatation of the pupils which do not respond to light (sometimes also visual disturbances), and a considerable increase of the pulse rate. Subcutaneous injections of atropine (0.03-0.05 gm.) have the same effect; this, however, comes on within a few minutes and lasts only for one to three hours (see Fig. 10).

Aside from its rapid development acute bloating of the lung is distinguished by the fact that it either comes on as a concomitant affection in acute disease of the lungs, or during hard work or after a long railroad transport, and that it disappears again after amelioration of the primary disease or during rest. It must, however, not be forgotten that the factors mentioned may also suddenly increase the symptoms of chronic

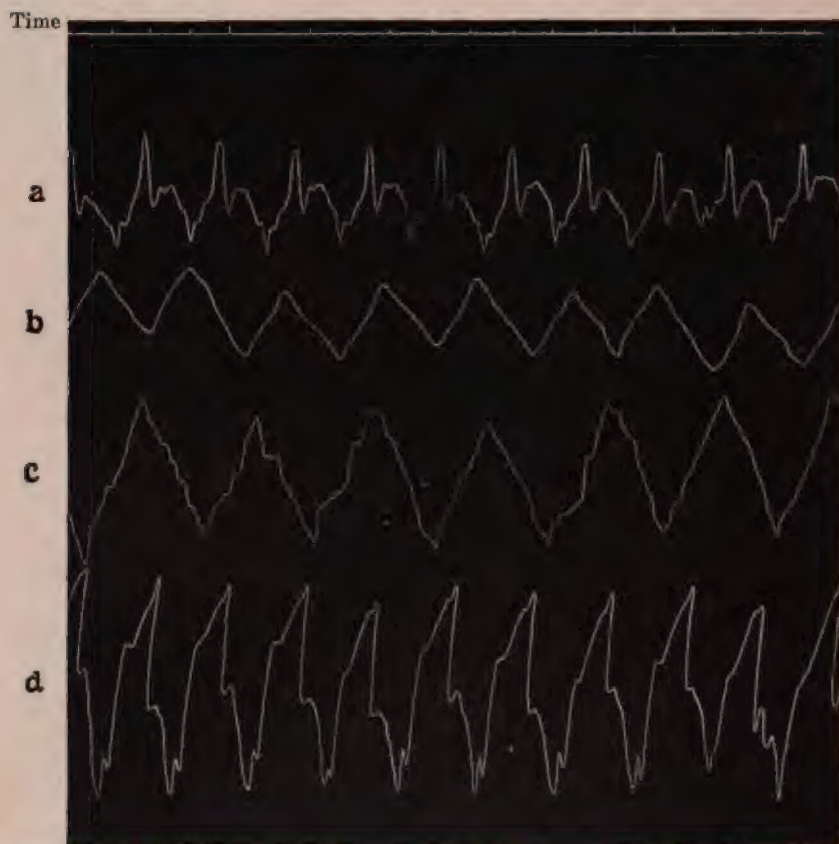


Fig. 10. *Pneumograph of a horse with pulmonary emphysema. a* before injection of atropine, (marked dyspnea with double expiration); *b* 15 minutes after the injection of 0.05 gm. of atropine, (dyspnea has disappeared); *c* 1¼ hours after the injection; *d* 17 hours after the injection, (dyspnea again very marked).

emphysema or that their discontinuation may much improve the clinical picture of emphysema. Interstitial emphysema is characterized by a rapidly increasing dyspnea, without the possibility of demonstrating a displacement of the boundaries of the lungs, and frequently with the development of subcutaneous emphysema. Pneumothorax is characterized by a sudden onset, by a metallic percussion sound and by similar breathing sounds.

**Prognosis.** It is impossible to prevent the progressive development of the disease; the prognosis therefore is unfavorable as to complete recovery. Since, however, the development goes on very slowly, the animals may occasionally be capable for years for walking and for pulling moderate work. The degree of the diminution of the ability to work may be ascertained by actual tests.

Every complication which appears during the course of the disease influences the prognosis unfavorably. This is especially true of bronchial catarrh which is seen so frequently as a complication; its complete cure will be much retarded in consequence of the insufficient exchange of gases. Cardiac weakness is of similar unfavorable prognostic significance.

**Treatment.** Good nutrition and appropriate work may be maintained upon in the absence of any specific curative treatment along the period of usefulness of the animals. Complications which may arise, especially bronchial catarrh, must be early treated (see page 61).

The systematic administration of arsenic (0.1-0.5 gm. per day) is generally indicated in affections of this kind which are characterized by an asthmatic condition. The beneficial effect of arsenic depends on its improvement of the nutrition. The Russians have found intratracheal injections of strychnine useful. Equally doubtful is the value of vergotinin (in teaspoonfuls), a mixture of veratrin, strychnine, ergotin and glycerin. It is injected daily 5-10 gm. of a 1% solution of atropine and produces only a temporary improvement (see page 97). Causes indicated in the use of eserine or chloride of barium are cases with emphysema, since in consequence of contraction of the muscle fibers of the bronchi these drugs may increase dyspnea and even lead to suffocation, as shown by the experiments of Raitsits.

**Literature.** Bouley, *Diet.*, 1878, V, 484.—Delafond, *Rec.*, 1837, 243.—Grüter, *Monatsh.*, 1905, 425.—Richter, *A. f. Tk.*, 1905, XXX, 576.—Schmidt, *Naturf. Vers.*, 1907.—Stömmmer, *D. Z. f. Tm.*, 1887, XIII, 93.

### Interstitial Emphysema of the Lungs. Emphysema pulmonum interstitiale.

Interstitial pulmonary emphysema consists in the accumulation of atmospheric air in the intra- and interlobular connective tissue of the lungs after solutions in the continuity of the alveolar walls.

**Pathiology.** Every considerable increase of the air pressure in the lungs, particularly if occurring suddenly, may directly



lead to rupture of the alveolar walls. The advent of the condition is therefore observed frequently after convulsive and forced attacks of cough. The following diseases may be indirect causes of the affection: acute catarrh and croupous inflammation of the finer bronchi; foreign bodies accidentally entering the air passages; improper drenching; pointed bodies which may directly injure the alveolar walls; over-exertion of the abdominal press in pulling a heavy load, in delivery, in vomiting or in efforts of animals which have met with an accident. Rupture of the alveoli may also be brought about by continuous bellowing, by forced expiration, in rapid running, in excitement, in railroad transportation, in falling or kicking (Arendt saw a case of this kind in a horse), after traumatic insults to the chest, etc. If the resisting power of the pulmonary tissue has been lessened, these forces may more easily lead to a solution of continuity, hence interstitial emphysema is often seen following alveolar emphysema.

According to Michels, Detmers and others, interstitial emphysema of the lungs occurs to an epizootic extent in some marshy parts of Holland and Belgium among cattle (*pneumosis bovum*). The cause of the disease has not yet been ascertained definitely, but it appears to stand in causal relation with frequent bronchial catarrhs due to exposure to cold. (Joest thinks that these cases are interstitial emphysema due to lung strongylosis of cattle, see page 72).

**Anatomical Changes.** Air bubbles varying in size from a nut to a fist, exceptionally also as large as a child's head, are found beneath the pleura and in the pulmonary parenchyma; the smaller bubbles are often present in large numbers and densely crowded. Otherwise the pulmonary tissue may be healthy or it may show the changes of a primary basic disease. The connective tissue of the mediastinum, of the chest aperture, of the neck and the subperitoneal tissue, may occasionally likewise contain air bubbles.

**Symptoms.** The air which has entered into the interstitial connective tissue soon compresses the neighboring alveoli; hence the respiratory surface becomes correspondingly diminished. In some cases the disease therefore sets in suddenly with dyspnea which progresses so rapidly that the animals sometimes are cyanotic and threatened by suffocation after a few hours. The percussion sound either remains normal or is accompanied by tympanitic accessory sounds. In the presence of large subpleural air bubbles it may be purely tympanitic. The respiratory sounds, both in expiration and in inspiration, are occasionally accompanied by crepitant and cracking noises. In some cases, particularly in cattle, subcutaneous emphysema is developed subsequently and moderate pressure on the tense, but otherwise healthy, skin elicits crepitation; the percussion sound is in these places tympanitic. The subcutaneous em-

physema may occasionally increase more and more, so that the whole body becomes swollen and disfigured. Subperitoneal emphysema can be felt with the hand introduced into the rectum.

The condition is sometimes rapidly aggravated, so that the animals suffocate within 1 to 2 days, while in other cases even an extensive air infiltration disappears and recovery finally takes place after a considerable period of time.

**Diagnosis.** The disease may be suspected if dyspnea has been preceded by the factors enumerated above; it can, however, only be diagnosticated beyond doubt when subcutaneous emphysema develops after the respiratory disturbances, and when other causes, such as injury to the upper air passages or to the esophagus, have been excluded. At its onset the disease may be confounded with hyperemia or edema of the lungs.

Acute edema of the glottis is differentiated by dyspnea associated with an inspiratory, whistling stenosis sound.

**Treatment.** To prevent if possible the further escape of air, complete rest must be enforced and cough, if present, must be alleviated with narcotics. Subcutaneous emphysema does not call for any special treatment, because the escaped air is gradually absorbed spontaneously, provided that the tears have been closed; slight massage may hasten the absorption. Puncture must be avoided on account of the danger of infection. Hasenkamp, however, produced rapid recovery in a sheep which had become emphysematous all over the body by making a number of small incisions into the previously cleansed skin.

**Literature.** Bouley, Dict., 1878, V, 482.—Delafond, Rec., 1832, 243.—Demeester, Ann., 1859, 342.—Hasenkamp, D. t. W., 1909, 472.—Littinger, Zündels Bericht., 1880-1881, 67.

## 5. Croupous Pneumonia; *Pneumonia crouposa*.

(*Pneumonia fibrinosa*; *Pneumonia lobaris*.)

Croupous pneumonia is an acute febrile disease with a typical course, associated with the occlusion of the alveoli of larger continuous parts of the lungs, with an exudate consisting of fibrin and blood corpuscles.

**Etiology.** Croupous pneumonia of domestic animals is caused without exception by an infection, whether occurring as a primary disease or whether it comes on as a complication in the course of specific infectious diseases. The infectious agents may be endowed with pathogenic properties from the start and may, after invading the animal's body, at once produce an inflammatory process. Croupous pneumonia of this type occurs as a localization of certain specific infectious diseases



(swine plague, horse influenza, contagious pleuropneumonia of cattle) and it appears more or less widespread according to the pathogenic characters of the particular causative microorganisms. Microorganisms which are morphologically and culturally identical with such disease producers are found not uncommonly as saprophytes in the air passages or in the environments of domestic animals without interfering with their health. If, however, the power of resistance of the organism as a whole or of the pulmonary tissue alone has been diminished by definite external influences or by other diseases, such microorganisms are enabled to penetrate into the organs of the body, to multiply in the lungs and to produce an inflammatory process in them. Their virulency may so increase that they are able to invade healthy animals without the auxiliary effect of external influences. It cannot be denied, on the other hand, that microorganisms living as saprophytes outside or inside of the animal organism may become pathogenic under special circumstances, and may be able to attack an animal the power of resistance of which has not been previously lessened. Such bacteria which have become pathogenic may lose their virulency in the diseased animal, or they may, on the contrary, retain it and spread the disease.

It has been customary for a long time to consider as a disease *sui generis*, and to call genuine pneumonia, that form of croupous inflammation of the lungs which appears, after certain external stimuli or without them, as a primary disease which does not, however, manifest a distinctly contagious type. This form has been separated from other types of pneumonia. Such a separation of diseases, which differ essentially only in the virulency of the infective agents concerned, does not, however, appear justified either from a scientific or from a practical standpoint, particularly since we are unable to determine at the onset the further behavior of the causative microorganism as to its virulency. Croupous pneumonia of man furnishes an analogous example; it appears sporadically at one time, endemically at other times, though the causative microorganism is the same under both conditions. We have no proof based upon sufficient bacteriologic investigations that there exists a separate clinical picture of croupous pneumonia occurring in any other way except by localization of well known infectious diseases.

Confusion as to the nature of croupous pneumonia has also been caused since the pathologic anatomical picture has been made the main basis of consideration, and therefore those cases have been designated as genuine croupous pneumonia in which a fibrinous exudate is found in the lungs, and where the specific infectious character of the disease cannot be recognized from the external clinical picture. It appears that the predisposition of the animal organism, to form a fibrinous exudate in the lungs after certain noxious stimuli, has not been



sufficiently considered; this predisposition, however, varies considerably according to species, age, nutrition, etc. Foreign bodies which have penetrated into the lungs, likewise food aspirated into the lungs of cattle, and also of other species of animals, may cause a croupous pneumonia; this, however, depends directly on the entrance of the foreign body and it does not lead to a typical clinical picture of croupous pneumonia. Some authors call all lobar processes croupous pneumonia, disregarding entirely the fact that certain bronchopneumonias may likewise assume the characteristics of a lobar process.

The occurrence of a genuine croupous pneumonia as a disease *sui generis* can therefore not be considered as an established fact. Croupous pneumonia of domestic animals develops in consequence of the localization of some known infectious disease in the lungs or in the course of a variety of internal diseases.

It has been a much contested question whether horses suffer from any other genuine croupous pneumonia except influenza. Several authors (Röll, Friedberger & Fröhner, Siedamgrotzky) believe in the occurrence of a genuine croupous pneumonia aside from influenza; the former, as a rule, occurs sporadically in horses and in other species of animals and is usually not contagious in nature. Other authors (Lustig, Schütz, Dieckerhoff, Cadéac) include all croupous pneumonia in horses under horse influenza. According to the view of the authors, so-called genuine croupous pneumonia in horses not infrequently shows such peculiarities in its appearance and course that the conclusion appears justified that these are simply cases of influenza (see Vol. I). One can observe in all extensive epidemics of influenza, that while a variable percentage of the horses of a stable shows typical symptoms of influenza, others, even many under the same conditions, suffer from a typical croupous pneumonia. It has also been observed a number of times that a horse, apparently suffering from a croupous pneumonia, may have infected its neighbor, or even all of the horses of the stable, with a disease which later on led to the typical picture of influenza. It is indeed impossible to draw an exact line between alleged genuine croupous pneumonia and influenza. The occurrence of sporadic cases of influenza is likewise not rare.

Croupous pneumonia in horses is seen sometimes as the sequel of external influences (cold, inunctions in skin diseases, irritating vapors, smoke, exertions, tying the head high, throwing for operations, contusion of the thorax). These were the very cases which were cited as proof of the existence of a genuine croupous pneumonia. It must, however, be claimed for the majority of such cases that the nature of the disease is influenzal, and that external factors, such as cold, which formerly used to be considered as the exclusive cause of the disease are only of importance as predisposing factors. These external in-

fluences act by lowering the resistance, to the influenza micro-organism, of the organism as a whole, or of the lungs in particular. It cannot, however, be denied that these external influences play a decided rôle which may be of such importance that the disease would never have occurred without them. These external causes may, in infected stables, lead to a so-called croupous pneumonia, while they will simply produce a catarrhal or interstitial pneumonia in non-infected stables, either after such external influences or without them; then there still exists the possibility that the influenza virus may have lived in the affected horse as a saprophyte and did not produce any noxious effect in the absence of a harmful predisposing factor.

Experimenters have not been able to produce croupous pneumonia in domestic animals. Dürck, who succeeded a few times in producing croupous pneumonia in small laboratory animals by considerable exposure to cold, found in all cases bacteria as the direct producers of the inflammatory process.

Secondary pneumonia is probably likewise due to the influenza bacillus and is sometimes seen after catarrhal influenza, epizootic laryngotracheal catarrh, strangles, hemorrhagic septicemia and purpura hemorrhagica. It does, however, usually not show the course of a typical pneumonia.

The occurrence of croupous pneumonia independently of pleuropneumonia in **cattle** is established beyond doubt; it develops usually after the invasion of bacillus bovisepiticus (see Vol. I). It is also commonly observed as a foreign body pneumonia after the entrance of foreign bodies from the air passages or from the forestomachs, although it does not show a typical course in these cases and usually takes the course of a catarrhal pneumonia.

The observation of Jensen, Buch and Krüger have shown beyond a doubt that hemorrhagic septicemia of cattle also occurs sporadically. Schütz found "ovoid" bacteria in the affected lungs in croupous pneumonia of cattle. Krüger demonstrated the presence of bipolar bacteria by inoculation experiments. Coulon and Olivier saw croupous pneumonia in cattle in wet valleys; Cagny in steers which were kept in the open during cold weather. These affections, however, could not be transmitted by application of the expressed lung juice to the skin denuded of its epithelium, or by subcutaneous application with a vaccination lancet. Nevertheless, the negative result does not exclude the possibility of pneumonia being caused by the bipolar bacterium, because subcutaneous inoculation is not always successful even when lymph of animals is used which undoubtedly suffer from the pectoral form of hemorrhagic septicemia.

**Hogs** suffer from the pneumonic form of swine-plague which often assumes a croupous character (see Vol. I). The cases of pneumonia observed by Bayer in Hungary probably belong to this type. Hemorrhagic septicemia which sometimes spreads from cattle to hogs may likewise form the basis of a croupous

pneumonia if it does not end in death rapidly. Since bipolar bacilli with the morphologic characteristics of *bacillus suisepiticus* occur in the buccal cavity, pharynx and nose of the hog, they may get into the bronchi with the feed, in greedy feeding or with accidentally inhaled foreign bodies, and may there cause a foreign body croupous pneumonia. The observations of Passerini and Wyssmann prove that anthrax in hogs may lead to a croupous pneumonia; the latter may form the exclusive localization of the anthrax infection.

The epizootic pneumonia which is rarely croupous in **sheep** is caused by *bacillus ovisepiticus* (see Vol. I); the infectious pneumonia of anatic goats (see Vol. I) is likewise due to bipolar bacteria; the cause of infectious pleuropneumonia of goats is unknown (see Vol. I).

According to older statements (Röll, Trasbot, Boissière, Renault) **dogs** were said to be frequently subjects of croupous pneumonia. These older statements, however, do not deserve much credence because the cases referred to were evidently distemper bronchopneumonias, which not infrequently involve whole pulmonary lobes. One may claim this with confidence, since the recent reports concerning croupous pneumonia of **carnivora** are very meager and the few cases published refer only to clinical observations. Distemper bronchopneumonia leads to only a partial formation of fibrinous exudate.

According to Friedberger & Fröhner **cats** suffer not infrequently from croupous pneumonia. Whether this disease is identical with the cat epizootic observed recently by Gärtner in Greifswald, cannot be decided definitely. The latter disease is, according to Gärtner, caused by the *bacillus pneumoniae felis*, a microorganism belonging to the group of bipolar bacteria; the affection consists of an extensive necrotizing pneumonia, principally of the posterior lobes; it is sometimes accompanied by a hemorrhagic, fibrinous pleurisy. This bacillus is probably identical with one described by Marx (*bac. pneumoniae tigris*) and found in a tiger dead from hemorrhagic pneumonia (Gärtner). Typical croupous pneumonia in the anatomical sense is also found in **carnivora** ill with glanders (Kitt).

**Rabbits** suffer from croupous pneumonia after an attack of contagious rhinitis (see page 15), or this affection represents the localization of such an invasion. Südmersen described a bacillus of the colon group as the cause of an enzootic pleuropneumonia of rabbits; Selter, one of the *bacillus bipolaris septicus* group (compare catarrhal pneumonia).

Croupous pneumonia in **fowls** is seen in slow cases of fowl cholera. Jowett saw an epizootic pneumopericarditis in turkeys, which was caused by the *bacillus bipolaris septicus*.

For details as to these affections due to bipolar bacilli the reader is referred to the first volume, dealing with infectious diseases.



**Susceptibility.** On account of the great prevalence of influenza, horses are most commonly affected with croupous pneumonia, as far as domestic animals are concerned. It is not merely relatively but absolutely frequent among horses and follows in frequency the colicky diseases. Younger, well nourished horses are most commonly affected; poorly nourished and overworked horses are less susceptible; young foals still less. Other domestic animals are affected more rarely or not at all by this form of pneumonia.

**Anatomical Changes.** Croupous pneumonia begins with an active hyperemia in a large continuous usually lower or anterior portion of one or both lungs (*stadium hyperaemiae*). The parts which are situated near the root of the lungs (*pulmonia centralis*) or the posterior, or still more rarely the upper portions are not often affected alone. Simultaneously with hyperemia there appear, not uncommonly, smaller or larger hemorrhagic foci. Extravasation of blood serum with white and red blood corpuscles occurs from the vessels into the alveoli and into the finest bronchioles, where the exudate coagulates at once, so that the affected pulmonary tissue becomes similar in consistency to hepatic tissue (*stadium hepatisationis*). The affected lung portion appears enlarged at this stage; its tissue is peculiarly tough and friable and it sinks in water. The cut surface, particularly on oblique illumination, appears uniformly finely granular and not juicy. Its color is at first red or brown-red (*st. hepatisationis rubrae*); afterward, however, fibrin and white corpuscles predominate in the exudate over the red blood corpuscles, the cut surface assumes a reddish-gray (*st. hepatisationis griseo-rubrae*), and later on a light gray color (*st. hepatisationis griseae*). Still later a yellowish color becomes more prominent in consequence of fatty degeneration (*st. hepatisationis flavae*).

While the inflammation sets in simultaneously in larger sections of the lungs, there are some deviations in the appearance of the cut surface; hence it frequently appears mottled (like granite), and red, gray and yellowish spots and streaks are found side by side. This so-called marbled appearance is also seen in croupous inflammation of cattle. However, the interstitial bands, if widened at all, are often only gelatinous, dilated lymph clefts not being visible, and the ground substance of the hepatized portions is much alike in color (see Vol. I).

After the exudate has become liquefied a reddish thick fluid mixed with fine air bubbles and fat droplets may be scraped off the cut surface, the consistency of the tissue has become softer. This stage of resolution leads to recovery after absorption and expectoration.

In the alveoli of the hepatized pulmonary tissue a reticulum may be seen under the microscope formed of fine threads of fibrin; its meshes are filled with white and red blood corpuscles, desquamated epithelia and granular detritus. Later on the alveoli contain only detritus and fat granules. The interstitial connective tissue shows an inflammatory infiltration with round cells and the fibers of the septa have been pushed apart by an edematous infiltration.

The connective tissue septa of the lungs are occasionally infiltrated, changed into gelatinous strips, several millimeters wide and dividing the cut surface into islands. Such strips are uniformly yellow and moist shining and are seen particularly in the lungs of cattle and hogs. The bronchi usually contain an abundance of a mucopurulent secretion; their mucosa is in a condition of catarrhal inflammation and croupous membranes are occasionally found in the smallest bronchioles.

Abscesses or necrotic foci are found in the hepatized portions or in those undergoing resolution in a portion of the fatal cases. The visceral pleura over the affected pulmonary portions is almost always lusterless, cloudy or rough, and occasionally infiltrated with small hemorrhages. Not uncommonly a fibrinous or serofibrinous pleurisy is found. The bronchial glands usually show acute swelling.

The post-mortem examination also shows secondary changes, such as cloudy swelling and sometimes fatty degeneration of the parenchymatous organs, especially of the myocardium.

**Symptoms.** Except in very rare cases the disease begins with the symptoms of a febrile condition with sudden onset. The appetite, and in ruminants also rumination are diminished; the former, however, is only rarely suppressed entirely. The patients stand listlessly and with drooping heads in front of the crib, smaller animals like to hide themselves and lie continually on the floor. Frequently one even observes at this stage cough and an accelerated respiration. The temperature rises to 40-41° C. and above within half a day and in some patients chills and muscular tremors occur. In older and debilitated animals the temperature does not show a considerable rise, but the other general symptoms reach an intense degree. If, for some reason or other, the animal already had fever, the onset of pneumonia is indicated by a sudden aggravation of the general condition and possibly by an additional rise in temperature.

After the fever has lasted from one-half to two, exceptionally also from 3 to 4 days, changes in the percussion and respiratory sounds become manifest, which can now be watched in modifications characteristic for the disease.

Percussion gives at first a somewhat dull sound, which assumes a tympanitic timbre usually on the second or third day, rarely earlier, and soon becomes purely tympanitic or it may also become gradually less intense. The change in sound usually shows itself at first in the region behind the elbow and spreads from there backward and upward to an extent which varies from case to case. As the alveoli become more and more consolidated, the tympanitic sound gradually changes into a dull tympanitic and later on into a very weak, dull sound. The area of dullness varies as to size and boundary



line. It usually extends backward from the elbow and reaches to the middle or even to the upper third of the thorax. Usually, although not always, the upper boundary line describes a curved line, with the convexity above, or descending backwards (see Fig. 11). It remains unchanged for three to five days, when the sound again assumes a tympanitic timbre, later on it becomes purely tympanitic and then gradually changes again into a normal, loud, non-tympanitic percussion sound. The low, dull sound only rarely changes to the normal without having first assumed a tympanitic timbre.

Deviations from the changes here described are observed. In affection of the deeper portions of the lungs the percussion



Fig. 11. Arched boundary of area of dullness in croupous pneumonia.

sound may either remain normal or not change until later on after the inflammation has reached the external strata.

If the consolidation of the upper boundary of the affected area is confined to the deeper strata, one hears above the upper boundary of dullness a tympanitic sound which may exist for several days. If the deeper layers are consolidated in such a manner that they nowhere touch the wall of the thorax, but are separated from the wall by considerable portions of healthy lung tissue, one hears only a tympanitic sound during the whole course of the disease, and this changes into a normal percussion sound during the stage of resolution. Deviations as to the seat of the area of dullness also are not



so very uncommon. The original change in sound over the lower portion of the thorax may gradually extend up to the vertebral column, or the change in intensity or timbre of the sound may occur exclusively on the posterior or upper pulmonary boundary and then may change in a manner already indicated (see Fig. 12).

During the initial stage of the disease auscultation reveals intensified and rough vesicular breathing on account of the swelling of the bronchial mucosa and of the accelerated respiration; even then high and crepitant râles may, however, be heard, especially during inspiration, indicating the presence of a serous, thick exudate in the alveoli and bronchioles. When



Fig. 12. Croupous pneumonia with an atypical location. *a*, area of dullness over the most posterior and upper portions of the lungs; *b*, area of a high, and *c*, area of a low tympanitic sound.

A tympanitic or weak percussion sound is audible, bronchial respiratory sounds are usually heard, and frequently also metallic râles. If the area of dullness becomes larger and the lumen of the smaller bronchi also becomes filled with an exudate there is usually an absence of all breathing sounds. Even in these cases, however, one usually hears bronchial breathing at the boundary of the area of dullness and further upward, because the hepatization of the lung reaches higher up in the deeper than in the more superficial portions of the lungs and bronchial breathing is conducted through the overlying, air-containing layers and is heard on auscultation, while percussion makes the air column in the outer strata vibrate; hence there is no dullness.





marked in horses. Cough is present from the start; it occurs, however, only at long intervals or after external stimuli, for instance, after percussion. In the stage of hepatization it appears painful, hence less violent, even very feeble, but it becomes markedly easy and moist with the advent of resolution.

The course of the fever is usually very characteristic for the disease in those cases which run their course without complications.

The temperature rises on the first day to 39.5°-41° C. and remains high with slight variations during the next days (febris continua—see Figs. 13 and 14). Toward the end of

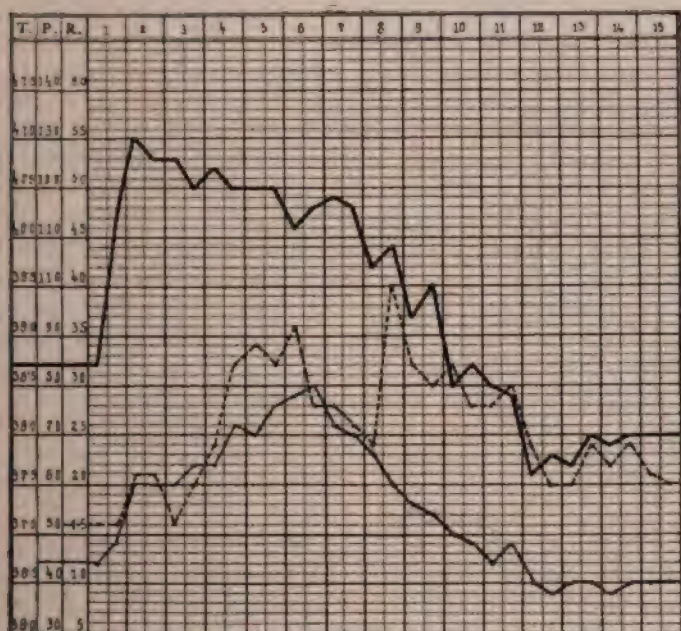


Fig. 14. Fever curve in croupous pneumonia of a horse. Lysis. Gradual increase of the frequency of the pulse and respiration with the approach of the stage of resolution.

The stage of hepatization, i. e., toward the end of the first week, the temperature either falls rapidly within  $1\frac{1}{2}$  to  $1\frac{1}{2}$  days (crisis—Fig. 13) or gradually, with remissions in the morning and exacerbations in the evening, so that the animal is free from fever only after 2 to 5 days (lysis—Fig. 14). The fall in temperature is usually preceded by a change of the dull and low percussion sound into a tympanitic or loud sound.

The temperature, which has fallen to normal or which is falling rapidly, may, in exceptional cases, rise again on the following day; it remains, however, high only for two days and then again goes down (perturbatio critica). Exceptionally the temperature may fall below normal, occasionally down to 36° C.; this occurrence, however, is like-



wise of short duration. The authors have seen intermittent fever in horses, as a rule, in combination with other severe symptoms.

The heart's action does not go parallel with the fever; there is, on the contrary, a certain antagonism between the fever curve and that of the pulse beat. The latter is only moderately accelerated in the beginning, in spite of the high fever (in horses up to 50 per minute) and the pulse is tense and full. The contractions of the heart become, however, more frequent in the latter course of the disease, and at the same time less strong; the pulse becomes empty and small (Figs. 13 and 14). This cardiac disturbance becomes the more marked the larger the affected lung territory and the more intense the infection. In the less severe cases the heart's action becomes again normal as soon as the stage of resolution is completed; on the other hand, there may be arrhythmia or allorhythmia of the heart; it is, however, usually of no significance even if it still exists during the stage of convalescence.

The general condition and with it the appetite of the animals varies a good deal from case to case. In milder cases the animals may ingest a normal amount of feed, though more slowly, during the whole course of the disease; others may refuse feed entirely during the course of the fever, and may show a considerable degree of listlessness and prostration. Most cases show at the onset a more or less marked diminution of appetite, a symptom which will first attract the attention of the attendants. Sick horses usually remain standing during the whole course of the disease; ponies and the smaller domestic animals lie down a good deal; in the case of unilateral affection they lie on the diseased side in order to permit the free expansion to the healthy lung.

The amount of urine decreases rapidly during the stage of hepatization and increases rapidly during resolution. The specific gravity is high during hepatization and sinks rapidly during resolution. The quantity of chlorine is diminished during hepatization; the amount of phosphoric acid, of sulphuric acid and of nitrogen is increased. The conditions are reversed with the advent of resolution (urinary crisis; Siedamgrotzky & Hoffmeister and also Wissinger have demonstrated these conditions in horses).

Storch demonstrated a considerable increase of nitrogen excretion in pneumonia of horses; this is explainable by an increased decomposition of nitrogen and nuclein compounds of the animal body during the course of the disease.

Urticarial eruptions sometimes appear, but do not last longer than two days. An exanthematous eruption similar to that caused by distillery mash and extending over all four extremities, was observed by Fröhner in a horse. Moderate perspiration sometimes comes on with the advent of resolution and with a rapid fall of temperature; strong perspiration is seen only in severe cases shortly before death.

The blood of horses with pneumonia shows a moderate hypoleucocytosis at the onset or during the fever; it changes into a hyperleucocytosis during resolution. The increase in the number of white blood corpuscles is mainly due to an increase in neutrophile also in acidophile cells (Sturhan, Wiendieck, Meier, Franke, Gasse). The number of red blood corpuscles and the amount of hemoglobin are diminished in most cases of pneumonia during the course of the disease; the values are, however, materially influenced by the ingestion of water and feed. If water is refused for several days and a loss of water of the organism occurs in consequence, the number of red blood corpuscles and the amount of hemoglobin may be relatively increased, though absolutely decreased (Wiendieck, Wetzl).

Deviations from the picture of typical and mild cases of pneumonia are not rare even in the absence of complications, but in such cases it is not possible to ascertain the cause of the atypical course. In stables with many horses one frequently notices that, simultaneously with a number of typical cases of pneumonia, a few cases occur with a markedly short duration. There is a sudden febrile attack with more or less severe general symptoms, but simultaneously with an improvement of the general condition the temperature goes back to normal on the second or third day (see Fig. 15). The thorax presents the signs of infiltration of the lungs (tympanitic, then dull sound, râles, sometimes indefinite breathing sounds), but they disappear after one to two days (pneumonia ephemera sive abortiva).

There are, on the other hand, cases in which the inflammatory process is confined to the deeper portions of the lungs which are not accessible to our methods of external examination (pneumonia centralis). These cases present for several days a high, continuous fever, although there are no demonstrable signs on the part of the lungs, and the patient may recover from an attack which has possibly lasted over a week, physical examination of the lungs having never furnished any positive data. However, one may reasonably suspect the presence of a croupous pneumonia from difficulties in respiration, from a saffron-colored nasal discharge which may be present, and from a typical fever curve.

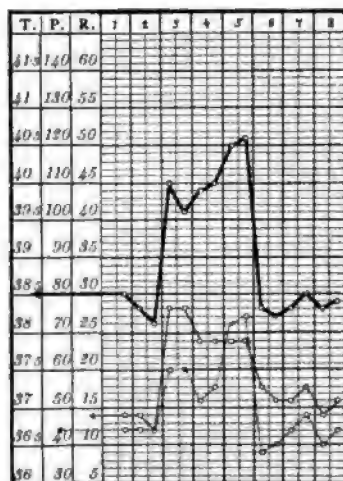


Fig. 15. Fever curve in croupous pneumonia of short duration in the horse.

Fairly frequent are those cases in which the inflammatory infiltration spreads from the pulmonary portions that are first affected to parts situated posteriorly and upward, or to the other lung (pn. progrediens). Under these circumstances the disease extends over a longer period, occasionally over several weeks, and the involvement of new portions of lung is indicated by elevations of temperature.

Recurrences occur exceptionally in cases in which the process has subsided, the fever disappeared and resolution taken place. In such cases the animal is, after a few days, again attacked by fever and the signs of pulmonary consolidation reappear at the place where they were found originally, or at another portion of the lungs (pn. recidivans).

The symptoms of croupous pneumonia vary also according to different **species of animals**. Horses present the picture described above. Elevation of temperature does not occur as rapidly in cattle as it does in horses; the acceleration of respiration is, however, more marked in the former than in the latter. Cattle frequently present a weak, painful cough which can easily be brought on artificially. Saffron-yellow or rust-colored nasal discharge is absent, but one may observe a whitish mucoid nasal secretion. Bronchial breathing is heard more rarely than in horses; vesicular breathing over the affected portions is more commonly weak or completely absent. The duration of the disease is longer; it lasts from one to three weeks, until resolution occurs in cases of favorable termination.

According to Gaertner's observation, during an epizootic among cats, these animals frequently sneeze and have an abundant nasal discharge, which is at first seromucoid and becomes purulent within two to three days. Masses of pus fill the nares, they move backward and forward during respiration, dry into crusts, often close up the nose and compel the animals to breathe through the mouth. The respiration is forced, often pumping, accelerated and accompanied by loud sniffing and snoring. Intense conjunctivitis is always present; it is at first serous then purulent. A weak, hollow and painful cough is heard in all cases. The temperature ranges between 39.5 and 41.6° C. The animals are at first depressed and morose, later on apathetic.

The affected animals usually die within three to six days, although exceptionally the disease may be prolonged to three weeks and more, and may then pass into recovery.

**Complications** are comparatively frequent in the course of croupous pneumonia. The degeneration of the myocardium plays an important rôle in the prognosis of the disease. A certain degree of cardiac disturbance forms a part of the typical clinical picture of pneumonia; these disturbances, however, assume a threatening character only in case of very grave infection or in very extensive spreading of the inflammatory



process. Cardiac weakness manifests itself in a pounding apex beat and in a weak to filiform pulse which may be increased to double the normal number or more per minute. There is then also collapse, cyanosis, the veins are over-distended and a venous pulse is noted. These circulatory disturbances are caused partially by the weakening effect of bacterial toxins on the vessel walls.

Another very dangerous complication is suppuration or gangrene of the pulmonary tissue, particularly in horses. The advent of this complication is sometimes announced by a chill and by elevation of the temperature which may even have been falling; then there is continuous or remittent fever to the end. Disintegration of pulmonary tissue is announced unmistakably by the onset of signs of cavity-formation (tympanitic sound persisting long in a circumscribed place, possibly associated with metallic sounds and amphoric breathing), and by the appearance of a disagreeable sweetish smell of the exhaled air. The symptoms of septicemia likewise become manifest. Under these conditions improvement occurs only very exceptionally. (Abscesses of the lung may break into a bronchus and may then heal completely.—Johns.)

Pleurisy is seen comparatively frequently; its occurrence as a complication is, however, quite variable. Tenderness of portions of the thoracic wall situated over the affected pulmonary areas, in connection with considerable acceleration of the pulse (Fig. 16), sometimes alone suggest the occurrence of a complicating pleurisy. Friction sounds are heard frequently or an abundant fluid exudate is formed in the thoracic cavity. The signs of pneumothorax (metallic sound, splashing, sudden respiratory difficulties) may occur in gangrene of the

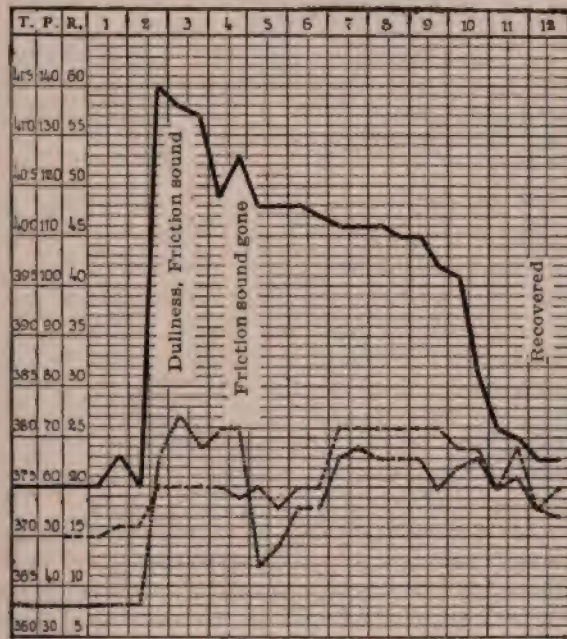


Fig. 16. Fever curve in croupous pneumonia with co-existing fibrinous pleurisy in a horse. Increased pulse frequency from the beginning of the disease.



lungs. Fibrinous or serofibrinous pericarditis is observed more rarely and still more rarely acute verrucous endocarditis.

Icterus is seen frequently in horses and is usually due to a simultaneous gastro-intestinal catarrh or to a hemoglobinemia from an influenzal or septic infection. Parenchymatous degeneration of the kidneys or acute parenchymatous nephritis are very common. They are announced by slight albuminuria which usually disappears with the fever. Acute diffuse nephritis is rare, it leads to profound albuminuria and to the appearance of renal tube-casts; renal epithelia and occasionally also degenerated red blood corpuscles are seen. Albuminuria is of grave significance, since it points to grave infection which is liable to lead to degenerative changes also in other organs, preferably in the heart.

Rare complications are: acute tendovaginitis and arthritis of the extremities, laminitis in the horse, iritis, acute meningitis and encephalitis. In horses pneumonia is sometimes followed by purpura hemorrhagica.

**Course.** Croupous pneumonia is one of those diseases which run a very typical course. The stages of inflammatory congestion, hepatization and resolution follow each other regularly. In horses pneumonia usually reaches its climax toward the end of the first week, i. e., on the fifth to seventh day; then all morbid symptoms usually disappear rapidly within one day, sometimes a little more slowly, so that the duration is at the utmost two weeks in all. The period of convalescence corresponds to the intensity of the attack and usually leads to complete recovery. In other animals the course of pneumonia varies more or less (see page 114).

Deviations from the normal course occur especially in cattle and swine, although they are not rare in horses. The sequence of the various stages remains the same, but the whole course may be of shorter or on the contrary of longer duration. Pneumonias caused by bacillus bipolaris or by external influences rarely take a typical course and those caused by the bacillus named (in cattle, hogs and sheep) may lead, within a few days, to a fatal termination. Complications which may develop also produce more or less marked deviations from the typical course and sometimes stand so much in the foreground that the clinical picture of pneumonia becomes completely clouded.

A fatal issue in consequence of suffocation may occur in very severe cases with extensive consolidation. Another cause of death may be grave degeneration of the myocardium. Cases with very severe infection lead to early and rapidly increasing prostration; usually toward the end of the stage of hepatization, the respiration becomes much accelerated and very forced, the heart is rapid and thumping, the pulse small, filiform, finally imperceptible; the mucosae become livid; there is finally a

general perspiration and the animals fall to the floor. In the meantime symptoms of pulmonary edema have developed. Suppuration and gangrene lead to death in other cases, after pyemic and septicemic symptoms have made their appearance. An edema of the glottis may sometimes produce death by suffocation.

A considerable number of cases of pneumonia is followed by a serous pleurisy; this must be suspected if dullness persists obstinately or even increases in the lower portions of the thorax and if more or less remittent fever is present. Severe pleurisy may cause adhesions of the pleura and this may lead to an asthmatic condition.

Chronic induration of the lungs not infrequently persists. This is indicated by a long drawn out stage of resolution, some difficulties of respiration persist; there may be moderate fever and the dullness does not clear up completely. A similar chronic process is sometimes developed in the neighborhood of a gangrenous focus, which may, even after weeks, produce an acute inflammation and cause death. A chronic pulmonary induration following upon acute pneumonic symptoms appears in horses and cattle tolerably frequently only in certain years, while in other years pneumonia rarely leads to this complication. The physical changes pointing to induration of the lungs disappear anyhow in a considerable number of cases 3 to 4 weeks after the fever has disappeared and the animals may be considered completely cured from a clinical standpoint.

In 0.4-2.8% of the cases occurring in the Prussian army the animals which had recovered presented after a few weeks the symptoms of paralysis of the larynx (q. v.). (Twenty-four [77%] of thirty-one thoroughbred horses of a stud, which had suffered from pneumonia, subsequently developed paralysis of the recurrent nerve [Plósz]).

**Diagnosis.** The typical form of croupous pneumonia can easily be diagnosticated in all species of animals. The sudden onset, a fever which is continuous for several days and falls either rapidly or gradually, the regular sequence of changes of the signs elicited by auscultation and percussion, recovery generally occurring in the second week of the severe affection, form a clinical picture which cannot easily be confounded with that of any other disease. None of the enumerated symptoms is of course characteristic by itself alone. Most characteristic is a rust or saffron-colored nasal discharge; however, this valuable symptom is often absent, even in horses. In the very first stage of the disease the cause of the fever is, of course, not obvious unless a similar affection has been observed previously in other animals. Systematic temperature observations among the animals in one barn will permit the early recognition of new cases, because changes in the percussion sounds will reveal the nature of the disease on the day following the observation of fever.



Errors of diagnosis may occur in pneumonia during the stage of hepatization, if a diagnosis has to be made on the basis of a single examination or if the disease is complicated by concomitant affections. One must think in this respect of pleuritis with effusion. Contrary to what is found in pneumonia, dullness is, in pleurisy, always in the lowest portions of the thorax, in horses it is usually on both sides, the upper boundary is almost without exception a horizontal line; the resistance in the area of dullness is increased, breathing sounds cannot be heard at all or only along the upper boundary; the disease comes on slowly, fever is usually less high, the course of fever irregular, breathing first of the abdominal type and later on very forced; dullness decreases or disappears after animal has been laid down and vesicular breathing takes its place; in smaller animals the dullness changes with changes in position in such a manner that it always appears in the lowest place. When pleurisy and pneumonia are present simultaneously some difficulty as to differential diagnosis prevails because the consolidated lung as well as the pleural exudate both cause dullness, while breathing sounds are often absent over the consolidated portions of the lungs. Edematous infiltrations formed on the lower thorax or lower abdomen may give some clue to the existence of pleurisy along with pneumonia. In doubtful cases an exploratory puncture should be made with a hypodermic syringe. The procedure is perfectly harmless even in the presence of an exclusive pneumonia. Only a positive result of the exploratory puncture can be utilized for diagnosis, because puncture may be negative even in the presence of pleuritic effusion.

Catarrhal pneumonia may furnish similar physical findings as croupous pneumonia if a number of small catarrhal foci have become confluent and have formed one larger consolidated area. However, bronchial pneumonia usually follows an extensive bronchial catarrh; it progresses slowly and lacks a typical course. One should never forget that adult horses usually suffer from croupous pneumonia, young foals and dogs exclusively, or at least preferably, from catarrhal pneumonia. The differential diagnosis from contagious pleuropneumonia in cattle is very important. During the stage of hepatization the findings may be identical in the two diseases; hence a reliable diagnosis can then only be made under consideration of the course. Contagious pleuropneumonia comes on gradually in contradistinction to the sudden onset and cyclical course of pneumonia, and cases with a comparatively rapid course last several weeks. The possibility that the contagious disease may be imported must always be considered. Inflammatory edema at the entrance of the larynx or in other parts of the body in the same or in other animals, the simultaneous occurrence of enteritis and the rapid fatal termination, point to a septic origin of pneumonia.

Foreign body pneumonia may be confounded with croupous pneumonia. It is, however, usually distinguished by a slower development. Fever is often absent at the onset or is very insignificant, bronchial breathing is likewise missed. After the entrance of foreign bodies from the stomach into the lungs, changes of the percussion sounds and respiratory sounds are noticed at first in the region of the diaphragm, and disturbances of digestion generally precede the affection of the lung.

In animals which are examined only after the fall in temperature, chronic inflammatory process, tumors, etc., must be excluded on the basis of a polyuria present, or of anamnestic data, and with the aid of an observation extending over several days.

In horses tympanitic sounds in the neighborhood of the lower pulmonary boundary may also be referable to the colon; this can, however, be recognized easily because a tympanitic sound of the same pitch can be elicited also beyond the pulmonary boundary over the abdominal wall or even below the costal arch.

**Prognosis.** The stronger the animal has been before the pneumonic attack came on and the more the latter conforms to the classical type the better is usually the prognosis. The course of the fever is of the greatest prognostic importance. If the temperature curve falls rapidly or gradually after a continuous fever of several days, a typical—that is, a favorable—course may be expected. Fever that has lasted over a week and has perhaps risen above  $41^{\circ}$  C. causes anxiety. A continuous, though not high fever, during or after the stage of resolution, points to retarded or incomplete absorption of the exudate or to the development of a secondary pleurisy.

The behavior of the pulse is of special importance. If, in the absence of a complicating pleurisy the number of pulse beats has risen to twice the normal, the pulse being at the same time weak, the prognosis is fairly unfavorable. It is also necessary to consider the extent of the inflammatory infiltration; the chances for recovery decrease in direct proportion to the size of the consolidated territory. Hence, bilateral pneumonia is more serious than a unilateral affection. Deviations from the usual localization have a similar unfavorable prognostic significance. Central pneumonia and the involvement of the upper parts near the vertebral column show a higher mortality.

The age and nutrition of the sick animals must be considered. In very old or debilitated animals a slowly developing pneumonia is observed, with moderate elevation of temperature, yet with marked prostration (so-called asthenic or adynamic type), which is always very unfavorable in nature and which generally takes a fatal issue.

Every complication diminishes the chances for recovery

more or less; particularly unfavorable are: pleurisy with abundant effusion, pericarditis, diffuse nephritis; to a lesser degree fibrinous pleurisy or intestinal catarrh. Symptoms of gangrene or suppuration of the pulmonary tissue point almost with certainty to an early lethal issue.

It must finally be considered that sequelæ may come on which will materially reduce the value of the animal. Since the advent of such sequelæ cannot be foreseen in the milder cases, the prognosis must always be guarded until the lung affection has entirely disappeared.

The mortality varies in horses between 0-20%; it is usually between 10-15%; in milder enzootics much less. In cattle the mortality varies considerably more; sometimes all cases get well (Strerath, Coulon & Olivier, Gotteswinter); at other times a mortality of 40% has been observed (Krüger) and even more (Guillebeau & Hess).

**Treatment.** The establishment of the most favorable hygienic conditions, especially sufficient ventilation of the barn or sojourn in the open, is of the greatest importance in influencing the course and termination of the disease. Working animals must at once be taken off work, to be kept in a moderately cool, well ventilated place or in the open air during favorable weather, but they must be protected against rain and wind. Where there are larger numbers of horses, turning them out into a yard will influence those that are affected favorably and prevent the spread of the disease. Weak large animals, especially horses, should be placed in slings. They should be permitted to lie down only at intervals and not more than two hours at a time. Rubbing of the body is beneficial and invigorating to the patients.

Since in acute febrile diseases the animals usually eat only juicy, dainty feed, herbivora should have fresh green feed, or if this cannot be had, good fresh hay, bran or flour mash perhaps mixed with some grains, also roots, beets, carrots, etc. If sick horses appear to prefer oats they should have them, mixed however with chopped vegetable roots. Carnivora should receive fresh milk, finely chopped boiled or roast meat, also strong meat-soups with one or more eggs. All food should be given in small but often repeated rations. The animals should frequently be offered fresh, but not too cold, water, also during the night, since the animals, and particularly the horses, are too weak to get to the suspended or otherwise placed water vessels. It is also advisable to clean the mouths of the sick animals with pure water before offering them food. They often take it then even if they appear previously to have no appetite. Where aspiration is threatening on account of disturbances of deglutition, food and water may be withdrawn, and moderately or well nourished animals may be made to fast for a few days. Later on, however, these patients and



those which are weak or emaciated must be nourished artificially (see page 123). The same procedure must be instituted with patients who entirely refuse to eat. Where water by mouth has to be withheld completely, water injections per rectum must be resorted to.

Drugs can be dispensed with in all cases with a typical course. If the pulse varies only slightly from the normal, alcohol is indicated in small, often repeated doses (ordinary spirits), larger animals 25-50 gm., sheep, goats, swine 10-20 gm., carnivora 1-2 gm.; wine  $\frac{1}{2}$ -1 quart or teaspoon doses, cognac, carnivora  $\frac{1}{2}$  teaspoon. Sulphuric ether (10 gm., 5 gm., or 0.25-1.0 pro dosi) may likewise be administered. If the number of pulse beats is increased to more than twice the rate, and also if they are weak, cardio- and vasotonic drugs are indicated. The most serviceable of these drugs is camphor, as ol. camph. according to Fröhner in tolerably large doses (for large animals 20-50 gm., for smaller animals 4-10 gm., for carnivora, 1-2 subcutaneously in one dose). The same results may be had with the much cheaper ol. camph. syntheticum (Fröhner) used in similar doses. Caffeine (for large animals 6-8 gm., for small animals 0.5 to 1 gm., subcutaneously every six hours) is likewise beneficial. (Gmeiner claims to have shortened the course of cases of pneumonia by the use of subcutaneous injections of caffeine.) Strophantinum purissimum Merck (horses 0.003 gm.) recommended by Regenbogen by subcutaneous injections in cardiac affections has not proved beneficial in the authors' cases. It acts much better by intravenous injection (for horses up to 0.015 gm.—Dorn, authors' own observation). For the purpose of subcutaneous injection strophantine must be dissolved in enough water (6-10 gm.) in order to avoid necrosis of the skin (Fröhner); however, even if applied in this dilution, a painful infiltration at the place of injection, which lasts several days, cannot be avoided. Such swellings are likewise frequently seen after injections of caffeine. Digitalis preparations to be used are: pulverized leaves of digitalis (for large animals 2-5 gm., for smaller animals 0.5-1.0 gm., for carnivora 0.05-0.3 for three to four days); also folia digitalis dialysata (for larger animals 5-15 cc. per os); further, digitalysatum Bürger (small animals 20-30 drops, subcutaneously). According to the investigations of Salvisberg, digitalis preparations are destroyed in the stomach of ruminants; hence the proper preparations must be given subcutaneously or intravenously. It must not be forgotten that digitalis has a tendency to produce abortion in pregnant cows. The general use of the excellent preparation digalen (horses 5-15 cc., cattle 20-30 cc., subcutaneously or intravenously) is frequently prohibited by its high price (Dorn); tincture of strophantus has been found unreliable (Regenbogen, Gmeiner).

In the face of threatening cardiac weakness the proper preparations of digitalis must be given intravenously because

then only can they produce the desired effect within a short time.

If disturbances of circulation have been brought about solely on account of a decreased tonicity of the arteries (see Vol. I) the effect of cardio-tonics may be augmented by intravenous injections of physiologic salt solution or of Ringer's solution (4-6 quarts for a horse). The intravenous injection of larger amounts of fluid may become dangerous in the presence of cardiac weakness and may then lead to fatal edema of the lungs (authors' own observation).

Fever does not, as a rule, require any special treatment, except in the presence of hyperpyrexia, when cold sponging, or irrigation or injections of cold water into the rectum may be indicated. However, cold douches should not be used during the cold season nor in very young or very debilitated animals. Of antipyretics the following may be used: antifebrin (15-30 gm. for large, 1-4 gm. for smaller animals, 0.1 to 1.0 gm. for carnivora pro dosi); phenacetin (the same doses) or antipyrin (the same doses). Lactophenin, which is much higher in price, may be given to smaller animals (0.5-1.0 pro dosi). With the exception of phenacetin or lactophenin these drugs must be used cautiously, since they may bring about collapse. The salicylates are indicated much less (acid. salicylicum, sodium salicylicum, salipyrin, salol, aspirin, dymal) since they often produce intestinal disturbances or irritate the kidneys. Talliamin (10 cc. intravenously) has not been effective in the treatment of influenza-pneumonia of horses, neither have inhalations of oxygen as recommended by Eberlein and Töpper (Pr. Vb. 1908, 6).

Venesection, which was formerly practiced universally, may bring temporary relief in the beginning; it does not, however, influence the course of the disease at all, and is by no means void of danger, on account of decrease of the tonicity of the arteries.

Delayed resorption may be stimulated by Priessnitz' applications and by diuretic drugs; of the latter we should use preferably acetate of sodium or potassium (25-30 gm. for large, 3-10 gm. for smaller animals, 0.2-1.0 gm. for carnivora, two to three times daily); diuretin, theozin, theophyllin (for carnivora daily three times 0.2-0.5 gm.); iodide of potash (for large animals 10-20 gm. pro die per os) iodipin (for large animals 30-50 gm. subcutaneously), fibrolysin (large animals 10 cc. every third day, dogs 2 cc. of the solution). The resorption of the exudate may be stimulated in protracted cases by puncture of the infiltrated lung with the hollow needle of an injection syringe; the procedure is similar to that used in exploratory puncture of the thoracic cavity.

The repeated inhalation of atomized disinfecting fluids (solution of corrosive sublimate 1:2000-4000, 2 to 3% solution of carbolic acid or creolin) may diminish putrefactive processes

in the air passages, but they cannot prevent putrefaction in the lung tissue itself after pulmonary gangrene has set in. Intra-tracheal injections of a 1% solution of carbolic acid (for horses 60-100 gm. at one time) or of a creosote or formol solution (see page 61) are better adapted for the treatment of pulmonary gangrene.

When pleuritis is present, it calls for special treatment (q. v.)

Stomatics (rheum, root of calamus, gentian, or their tinctures for smaller animals), artificial Carlsbad salt, hydrochloric acid with pepsin may be administered to stimulate appetite. Convalescent animals must be well nourished and not used for work until they have regained their full strength. Moderate exercise in the open is beneficial if the weather is favorable.

Since croupous pneumonia is usually a localization of some acute infectious disease, such prophylactic measures as are recommended for the particular infection, must be carried out.

**Artificial Feeding of Sick Animals.** The simplest and most practical method of artificial feeding of animals consists in the introduction of food stuffs into the stomach. It consists in its simplest form in pouring liquid food into the mouth from a thick-walled glass bottle or from an irrigator or funnel provided with a rubber tube, or, in smaller animals, from a spoon. Flour and bran gruels are well adapted for herbivora; milk, broth or flour soups, to which some glucose or eggs may have been added for carnivora. If difficulties of deglutition exist, and if attacks of cough come on this procedure must be abandoned and the liquid food must be introduced directly into the stomach by the aid of a stomach tube.

Artificial feeding per rectum becomes necessary in those cases in which there exist great tenderness of the pharynx, great excitement of the animal during the introduction of the stomach tube, obstruction of the esophagus, intense inflammatory processes of the stomach or small intestines, in short, in all cases in which the introduction of the stomach tube into the stomach becomes impossible or is contraindicated. Before food stuffs are introduced into the rectum, the fecal masses which are present must be removed manually or by irrigation with water one-quarter to one-half hour before the operation; a liquid food enema may then be introduced with an irrigator, thin mashy material with the aid of a rectal syringe. These substances must be warmed to body temperature; they must be introduced deep into the rectum, but without undue pressure. The early discharge of the nutritive material from the rectum must be prevented by pressing the tail to the anus, or by the addition of opium to the clysmas. It appears advisable to add to all nutritive enemas 1 to 2% of common salt, since this produces antiperistaltic movements, transports the food material higher up and also favors its general absorption. If nutritive enemas are used daily it becomes necessary to wash out the rectum once daily in order to remove decomposing remnants. The amount which may be introduced at one feeding, three to four times daily, is for small dogs and cats, 10 to 40 cc.; for large dogs, 100 to 200 cc.; for small ruminants, foals and calves, 200 to 400 cc.; for adult horses and cattle two to three quarts (Jacob). In considering the composition of the nutritive enemas one must never forget that the mucosa of the large intestine can only change starch and saccharose into glucose, but does not possess any other digestive properties. It possesses, however, considerable absorptive power; it may therefore absorb, aside from water, salts, and glucose, also peptone, albumoses and other albuminoid bodies and small amounts of emulsified fat. Dogs can however, only absorb 4 gm. of fat daily (Ornstein). The following may therefore be said concerning the composition of nutritive enemas: Best adapted, at least for dogs, are nutritive enemas of milk, starch, glucose, and raw eggs (Ornstein); the amount of glucose should not be more than 2%, of starch 8 to 10% of the total amount. This enema is prepared in shaking starch with cold water and pouring this emulsion into boiling water so as to produce a uniformly gelatinized starch mash, to this is added grape sugar which has been dissolved previously in hot water; finally two to three eggs stirred with a little water and common salt are slowly mixed with the cooled starch paste under constant stirring to insure a uniform mixture.



The artificial enemas which have been recommended by Leube for man may also be utilized; such are: peptone-milk enema (250 gm. milk, 60 gm. peptone); egg-milk enema (250 gm. milk, 3 eggs, 3 gm. common salt); starch-milk enema (60 gm. starch, 25 gm. milk); sugar enema (60 gm. glucose, 250 gm. milk); pancreas enema (50 to 100 gm. pancreas, 150 to 300 gm. milk, 30 to 45 gm. fat). A simple meat-pancreas enema is prepared by mixing finely chopped beef with finely chopped fat-free cattle or hog pancreas (3-1) to which mixture it is well to add some fat (30 to 40 gm. cod liver oil, previously emulsified with a few tablespoonfuls of 0.3% solution of carbonate of sodium); further, 6 to 10% of starch or a maximum of 2% glucose. In the treatment of valuable animals pancreatin may be used in place of fresh pancreatic tissue and the meat may be replaced by albumin preparations (peptone, meat-meal, roborat, tropon, kalodal). Ornstein's experiments have shown that dogs are able to absorb milk from the rectum, and also blood serum of other animals (up to 20%); however, serum should not be used for longer periods than ten days, since it may produce hypersensitiveness (anaphylaxis—see serum disease). The digestion and absorption in the rectum of herbivora has not yet been studied experimentally with reference to the exact metabolism.

Artificial feeding by subcutaneous injections of nutritive material must also sometimes be resorted to if disease of the large intestines prohibits the administration of food enemas, or if these are expelled at once. The excellent investigations of Ornstein in dogs have shown that 150 cc. of blood serum to which 10 to 20% glucose has been added will be absorbed and assimilated, almost completely protecting in this way 22% of body proteids against decomposition. It could also be shown that the intestinal mucosa is in some way concerned in the assimilation of proteids injected subcutaneously, and that the end-products of oxidation are different from those formed normally; the urine of animals fed by the parenteral route is characterized by a higher contents in calories. Liquid food is best incorporated in this way by an infusion apparatus, under low pressure and at body temperature. Heterogenous sera should only be used for a few days, in order to prevent the onset of anaphylactic symptoms. The use of homogenous serum is more to be recommended particularly in horses. According to Friedenthal & Lewandowsky, heterogenous sera lose their poisonous properties if heated for a considerable time at 58 to 68° C. Kalodal (phosphorous albumin) is considered non-poisonous on subcutaneous administration. Since fats are only absorbed in traces, their use is not to be recommended unless homogenous fats are used. Fluids which are to be injected subcutaneously must be sterilized; blood serum, if used, must be collected under aseptic precautions. Fats (oils, butter, lard) may be given in doses of 50 to 70 gm. to larger, and in doses of 10 to 15 gm. to small animals; these doses are injected in several places under the skin and the procedure is repeated four times so that the daily amount is equal to about 300 or 50 to 120. The amount of blood serum mixed with 10 to 20% solution of glucose or of glucose solution alone (10% glucose in physiologic salt solution) may be 200 gm. pro dosi in dogs, one liter in larger animals. Kalodal is injected into dogs in a 1% solution; 5 gm. of kalodal are dissolved in distilled water, the solution is sterilized and is then diluted with 500 gm. sterile physiologic salt solution.

For intravenous feeding, at best only grape sugar dissolved in physiologic salt solution could be used, but it can only be injected in such amounts as will be retained in the blood (the normal average amount of sugar in the blood is 1%).

In considering the importance of artificial feeding it must primarily be pointed out that artificial stomach feeding is the method which most nearly approaches physiologic conditions and which permits of the introduction of the largest number of calories. Large animals may be kept alive for weeks by this method, provided that the secretion of the digestive organs is not much below normal as is so often the case in grave acute infectious diseases. Rectal and parenteral artificial feeding can, at best, only diminish the disintegration of substances that are stored up in the organism. It is insufficient to keep otherwise starving animals, especially larger animals, alive for any length of time. With rectal artificial feeding one can introduce in dogs only 100 to 150 calories, and a healthy dog, weighing about 20 pounds, needs about 300 calories. By a proper combination of rectal and parenteral feeding smaller animals might possibly be kept in equilibrium for some time. In larger animals rectal and parenteral artificial feeding can only serve to retard malnutrition in diseases which do not last very long. The technical difficulties are also opposed to its use in general practice. In the rectal mucosa a catarrhal condition is easily produced if rectal enemas are administered daily, even if easily putrefactive substances, such as eggs, are not given for more than three days, and if glucose is given in a dilute, not too concentrated, solution. Abscess formation and cutaneous necrosis at the site of the application may occur after cutaneous injections in spite of all precautions; intravenous injections cannot

be administered several times a day, even in larger animals. Intravenous injections of glucose solution, 80 to 100 gm. in two to three liters of physiologic salt solution, can only be used occasionally to combat rapid physical weakness.

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## 6. Catarrhal Pneumonia. Pneumonia catarrhalis.

(*Bronchopneumonia, Pneumonia lobularis s. insularis.*)

Catarrhal pneumonia is an inflammation of individual lobules or groups of lobules of the lungs, leading to an occlusion of the alveoli with desquamated epithelia and a cellular exudate, which usually develops by the continuation of a bronchial catarrh into the corresponding lobules or simultaneously with bronchiolitis.

**Etiology.** Those factors which may cause bronchitis or bronchiolitis (capillary bronchitis, see page 54) may all cause catarrhal pneumonia, but there are special factors which are particularly prone to produce bronchopneumonia. The inhalation of very hot air, or air containing irritating gases, the entrance of foreign bodies (fluids or solids) into the air passages may cause bronchopneumonia, which may frequently terminate in gangrene, particularly if caused by foreign bodies. Foreign bodies generally get into the air passages by aspiration in swallowing, but they may also be inhaled in regular respiration. Wool threads (Dralle) and parts of nut cakes (Habicht) may in this manner be inhaled into the lungs of cattle, and if these animals are fed with cottonseed or ground-nut cakes there may be numerous cases of this kind. Bronchopneumonia seen in horses after chloroform narcosis also occurs in consequence of aspiration, if these animals receive solid food directly after the narcosis (Ducasse). The observations of Siedamgrotzky and



Johne concerning an enzootic bronchopneumonia of cattle in the neighborhood of Freiberg are highly interesting. This form of pneumonia developed in consequence of the inhalation of smelter-smoke containing arsenic and it predisposes very much to the development of tuberculosis.

According to Johne older horses not infrequently develop bronchopneumonia in consequence of long continued stay in the stable ("stable-pneumonia"), because a prolonged sojourn of this kind leads, through insufficient breathing, to an accumulation of the bronchial secretion with the subsequent decomposition of the collected masses; this again induces partial pulmonary emphysema, atelectasis and finally bronchopneumonia. Bechmann, however, considers stable-pneumonia as a form of croupous pneumonia (influenza).

Parasitic worms (see page 66) are frequently, moulds rarely, the cause of catarrhal pneumonia.

The great majority of cases of catarrhal pneumonia develop on the basis of an infection. The same organisms which cause croupous pneumonia may especially be active (Weichselbaum), particularly in very young, or on the contrary, in old debilitated animals.

The bacillus bipolaris septicus may cause bronchopneumonia, particularly in cattle. (According to Smith, the sporadic pneumonia of cattle is a bronchopneumonia caused by the bacillus bipolaris). Bronchopneumonias are also often caused by bacillus pyogenes. However, this bacillus probably invades the lungs only secondarily and is able to display pathogenic properties there only if the bronchial mucosa has become predisposed at the time of the invasion or previous to it (Holth, Reisinger) in consequence of non-specific irritation (cold, inhalation of dust, congestion of the lungs). Martens, Berstel, Schmidt and Lewek have described a bronchopneumonia occurring in Germany, either sporadically or enzootically, as an infectious disease sui generis, beginning with the symptoms of catarrh of the upper respiratory passages (see page 45) and leading to bronchopneumonia in a portion of the cases. It can not yet be decided what is the relation between this affection and bronchopneumonia caused by the bacillus pyogenes, which was described more fully by Reisinger. Lewek found Gram-positive bacilli or Gram-positive cocci or both in the affected lungs. Prolonged transportation on railroad cars or on board ship, unhygienic conditions and colds play a predisposing rôle. The disease is particularly common in market cattle.

Bronchopneumonia caused by the bacillus pyogenes occurs also in hogs (Borges). The etiology of contagious bronchopneumonia observed by Röbert and Deich in dogs of every age is entirely unknown. Rabbits frequently suffer from bronchopneumonia either in connection with contagious rhinitis or as a primary affection which is, however, caused by the same infectious virus (infectious pneumonia of rabbits). A bacillus of the coli group, described by Südmersen (see page 105) likewise causes epizootic bronchopneumonia and catarrhal disease of



the air passages in rabbits, so that the clinical picture is similar to that of contagious rhinitis. Frosch & Bierbaum found a specific microorganism, the bacillus septicemix anserum exudativæ as the cause of an epizootic purulent bronchopneumonia in geese.

Bronchopneumonia often occurs secondarily, especially in young animals, in the course of acute infectious diseases. For instance in distemper in dogs, in hemorrhagic septicemia in sheep which was also known as sheep glanders, in some cases of malignant catarrhal fever, in foot-and-mouth disease, in sheep-pox, purpura hemorrhagica, hog cholera; more rarely in chicken cholera. Tubercle and glanders bacilli, also ray-fungi may be the cause of catarrhal pneumonia. (For chronic catarrhal pneumonia occurring in South Africa and there called "Jagziekte," or "Hartslagziekte," see Vol. I).

**Predisposition.** Predisposing factors often play an important rôle. Such predisposing factors are: very early, or on the contrary, very advanced age, cold, debility of the organism as it occurs in anemia, in grave nutritive disturbances, in rachitis, etc. These predisposing causes may, however, also be absent in infectious catarrhal pneumonias.

**Anatomical Changes.** The inflammation is, as a rule, confined to certain foci, to individual lobules or groups of lobules; hence there are at first smaller, later on larger pneumonic areas, which may become confluent and form large consolidated territories. These consolidated areas are usually in the neighborhood of the lower and anterior pulmonary margin. They are solid; not as firm, however, as in croupous pneumonia. The cut surface is at first flesh-colored, later on more and more grayish-red, and, when the course of the affection is very slow, yellowish or grayish-white and then dry. The cut surface is not granular; aside from catarrhal, there are also seen atelectatic foci, which are a little darker, but airless, wedge-shaped, sunken in under the general surface and uniformly meat-like to the touch. Other portions are not uncommonly emphysematous and bloated. In cattle interstitial pulmonary emphysema frequently exists simultaneously. If the disease has lasted a long time one may see whitish or greenish purulent foci of various sizes in the consolidated areas, and in these Preisz' pseudotubercle bacillus, or bacillus necrophorus, in hogs and cattle the bacillus pyogenes are found occasionally.

Swollen, partly fatty-degenerated, desquamated epithelia, a variable number of erythrocytes and leucocytes, occasionally also threads of fibrin are seen in the alveoli of the catarrhal foci under the microscope. In bronchopneumonia of cattle fibrin is frequently found abundantly in the inflamed pulmonary portions. The alveolar vessels are dilated, the interalveolar tissue is edematous and infiltrated with round cells.

The bronchi, especially the smaller ones, always contain an abundant mucoid or purulent, occasionally more caseous exu-

date, which is discharged from the cut surface. The mucosa is reddened, loosened and swollen.

In consequence of serous and cellular infiltrations the interlobular connective tissue appears occasionally gelatinous or whitish; this is often the case in cattle, occasionally in hogs, and only exceptionally in other animals. The pleura frequently appears healthy, occasionally somewhat roughened, at times also covered by a fine fibrinous membrane. The bronchial lymph glands are in a condition of acute swelling.

**Symptoms.** Catarrhal pneumonia is usually preceded by the symptoms of bronchitis or bronchiolitis; frequently the former manifests itself simultaneously with the latter. In the former case an aggravation of the symptoms points to the advent of catarrhal pneumonia.

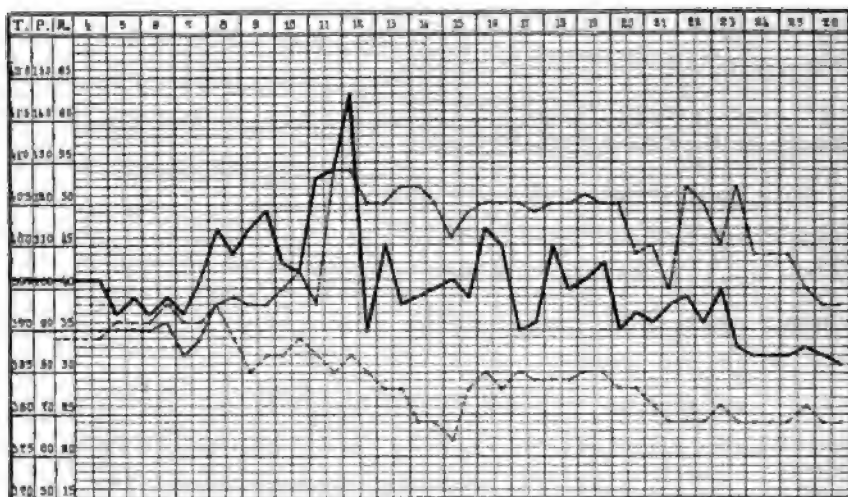


Fig. 17. Fever curve in catarrhal pneumonia of the dog, terminating in recovery.

When bronchiolitis is already present, an elevation of the temperature above 40° C. at once creates the suspicion that catarrhal pneumonia has developed following the primary disease. This may be held with almost absolute certainty if the patient is very young or very old. The fever does not show a typical course; it is of a remittent type and disappears gradually after recovery has set in (Figs. 17 and 18). Catarrhal pneumonia of adult animals may, for some time or even up to the end, run its course without fever and without other grave general symptoms.

The respiration is accelerated and made difficult in direct proportion to the extent of the inflammatory process; dogs, more rarely other animals, blow up their cheeks with every expiratory movement (so-called labial breathing). The animals cough from time to time or continually; the cough is usually

short, dull, frequently painful, and consequently the patients try to suppress it. One usually observes also an abundant nasal discharge.

Percussion produces pain and frequently excites cough. Careful, attentive percussion reveals dull or very low sounds in circumscribed places of the thorax. These changes are usually found above and along the lower pulmonary border, only exceptionally in the middle or upper third of the thorax. The percussion sound may have a tympanitic timbre at the boundary of the area of dullness or in other places. Small animals sometimes present the cracked pot sounds.

Auscultation reveals a variety of catarrhal sounds, sometimes circumscribed places, as a rule, now, at times over, the whole of the thorax. Sounding (metallic) rales have a special significance if they are present. In exceptional cases, if the lungs have become consolidated to a larger extent, while the lumina of the larger bronchi have remained open, one hears bronchial breathing in the area of dullness; as a rule, however, the breathing sounds are more or less weakened, because the bronchi are stretched by the catarrhal secretion.

The pulse is accelerated, at first quite strong; later on it becomes weakened.

The general condition suffers materially in most animals, both on account of the fever, and on account of the respiratory difficulties and the tormenting attacks of cough. Small animals usually rest on the sternum. The appetite is diminished or entirely suppressed; sucking animals do not go to the mother.

Infectious bronchopneumonia of cattle follows, as stated, after infectious catarrh of the upper respiratory passages (see page 45) (according to Schmidt, in 50% of the cases). Three to four days elapse, according to Lewek, before catarrhal pneumonia develops; catarrh of the upper respiratory passages may, however, assume a slow, insidious course and may bring about pneumonia at any time (Reisinger). If this is the case the fever comes on suddenly, and dullness and bronchial breathing can soon be demonstrated over the anterior and lower portions of

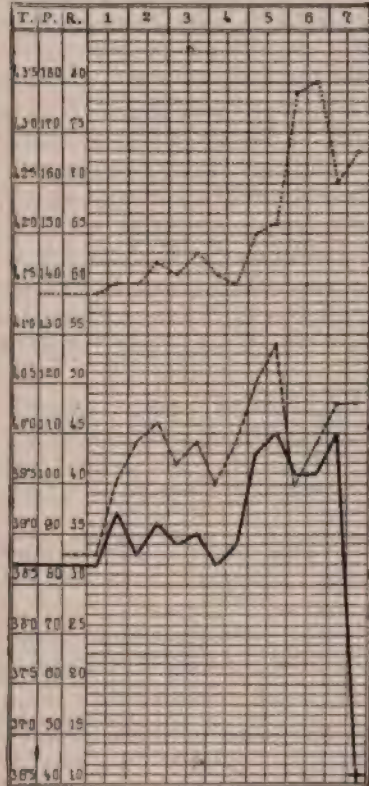


Fig. 18. Fever Curve in catarrhal pneumonia of the dog with terminal collapse temperature.



the thorax. The fever may, however, disappear again within a short time; the appetite may improve, yet the signs of pneumonic consolidation persist without influencing the general nutrition or the yield in milk. In this form the disease lasts about fourteen to eighteen days and reaches its climax about the tenth day; then the symptoms decrease and most animals recover completely (Lewek). Occasionally the course may be less favorable. In both forms interstitial pulmonary emphysema is not infrequently developed.

**Course.** Catarrhal pneumonia follows a very variable course, in accordance with the variety of its causes. It develops very rapidly in some cases and may have assumed a considerable extent within a few days; the development is slower in other cases and the course may extend over several weeks and even months. The fever subsides in favorable cases toward the end of the second or third week, or often even earlier; the cough becomes easier, moist, less frequent; the respiration less accelerated and forced, and complete recovery takes place. Renewed elevations of temperature may come on during the period of recovery; this is then due to the formation of new inflammatory foci.

In other cases the disease takes a fatal termination, and the animals die from suffocation, cardiac paralysis, exhaustion, intercurrent pleuritis or pericarditis, or from profuse diarrhea, occasionally also from pulmonary gangrene or in consequence of a general sepsis. Again, in other cases there may remain a chronic pulmonary induration; the animals then suffer in their nutrition and from respiratory difficulties. Young animals are retarded in development under these circumstances.

**Diagnosis.** The greatest difficulty is offered by the differential diagnosis between catarrhal pneumonia and bronchiolitis; a careful physical examination, however, often enables us to come to a definite conclusion. High fever, absence of vesicular breathing, and particularly dullness on percussion, speak in favor of catarrhal pneumonia. Catarrh of the bronchioles rarely exists for any length of time without the advent of catarrhal pneumonia. Croupous pneumonia can be distinguished by its much more rapid development, its acute and cyclical course, by the extensive and strong dullness, the bronchial breathing which is frequently present over a wide territory and finally by its often favorable termination. Bronchopneumonia in cattle can not be differentiated from pulmonary tuberculosis without any further study when it takes a somewhat protracted course. In contradistinction to pulmonary tuberculosis, bronchopneumonia does not lead to severe emaciation, even after severe local changes, and the catarrhal sounds remain, as a rule, confined to the anterior and inferior portions of the thorax.

**Prognosis.** The prognosis is the more unfavorable the younger or the older the animal. The poorer the state of nutri-

tion, and the higher the fever, the slighter are the chances for complete recovery. The course is particularly unfavorable if the catarrhal pulmonary infection comes on during an acute infectious disease. Röbert observed a mortality of 30% in epizootic bronchopneumonia of dogs; Deich, 50%; Schmidt reported 30% mortality in contagious pneumonia of market cattle.

**Treatment.** The hygienic environment and diet should be regulated according to the same principles as were laid down for croupous pneumonia (see page 120). Sucklings suffering from lack of appetite must be fed with freshly drawn milk or with boiled milk cooled down to body temperature; two to four eggs may be added; eggs can also be given raw or mixed with warm wine soups.

To improve the bronchial catarrh inhalations may be practiced (see page 7 and page 42); inhalation of oxygen might likewise be tried. Narcotics (see page 42) should be used only in the presence of tormenting, convulsive cough, and even then very cautiously. The regulation of the heart's action requires special attention; the principles are the same as those laid down for croupous pneumonia (see page 122). Priessnitz' applications to the thorax may be useful. If a complicating pleuritis, pericarditis or enteritis is present, they should be treated suitably.

**Prophylaxis.** If the disease is due to an infection, the animals which are still healthy should be removed to suitable quarters, preferably to some distant place; they ought to receive a change of diet and water and disinfection should be practiced repeatedly. Newly bought cattle should be isolated for one week (Lewek).

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**Pneumonia of Birds.** This is rare with the exception of pneumonomycosis. According to Zürn (Geflügelkrankheiten, 1885, 286), the symptoms are accelerated, short, whistling respiration through the open bill, tenderness and elevated temperature of the thorax, cough with the expectoration of tenacious, grayish-white, sometimes, or hemorrhagic saffron yellow masses, listlessness, lack of appetite.

The treatment is similar to that for bronchial catarrh or for pneumonomycosis.

**Atelectasis of the Lungs.** This occurs very frequently among domestic animals. Simader, who studied this affection in the Leipzig



slaughtering house, found 15% of the slaughtered young pigs affected; 50% of sucking pigs; 7% of young goats; sucking calves, 13%; young sheep, 16%. Atelectasis may be primary or congenital, or secondary and acquired. Congenital atelectasis is due to the fact that portions of the lung do not expand after birth and remain airless during extra-uterine life. Acquired atelectasis occurs if some bronchi become impervious (obstruction atelectasis) or if portions of the lung become pressed upon (compression atelectasis). In both cases the air is absorbed within a few hours and the portions affected become void of air. As a third form we may mention marantic atelectasis which also is due to a gradual absorption of the air out of the alveoli. Simader believes that atelectasis in food animals is a congenital affection.

The **cause** of congenital and marantic atelectasis is usually an atrophic condition of the respiratory muscles due to fatty degeneration, lack of development, or due to various diseases of infancy (dysentery, polyarthritis, rachitis, rheumatism); not uncommonly atelectasis can be referred to insufficient physical exercise if the animals are kept continually in the barn. Obstruction atelectasis usually occurs during bronchial catarrh, more rarely in new-born animals, in consequence of the obstruction of bronchioles by epithelial plugs, by mucus or by aspirated meconium. Compression atelectasis is usually caused by an accumulation of fluids or air in the pleural cavity, more rarely by voluminous tumors of the lungs or pleura.

**Anatomical Changes.** The seat of atelectasis is usually in the upper portions especially the anterior lobes of the lungs. The atelectatic foci appear browned, sunken in under the surface of the lungs and wedge-shaped if they are due to a bronchial obstruction. Congenital atelectasis is characterized by its typical location in the apices, by a normal appearance of all bronchi, or at least of those in the neighborhood of the atelectatic focus, and also by the fact that the pleural covering does not appear folded. If a portion of lung has not been expanded properly, hyperemia occurs and soon a serous infiltration of the atelectatic focus (splenization) develops; the epithelia then undergo fatty degeneration and become desquamated; mucoid material collects in the bronchioles and leucocytes wander into the interstitial connective tissue (desquamative catarrh). The tissue elements which have undergone fatty degeneration are absorbed and this is followed by marked atrophy which may progress to a degree that the two opposite leaves of the pleura finally touch (induratio).

There exist no observations concerning the clinical **symptoms**. It is, however, probable that in extensive atelectasis disturbances of respiration and physical changes in the thoracic sounds may be found similar to those encountered in catarrhal pneumonia. Congenital pulmonary atelectasis must always be suspected when animals are apparently still-born. Atelectatic foci of lesser extent do not cause any symptoms at all.

The **significance** of the lesion depends upon the underlying cause. Atelectasis is of a certain importance because it might be confounded with contagious bronchopneumonia of young animals and an inflammatory process may be established secondarily in the atelectatic focus (usually bronchopneumonia, exceptionally croupous pneumonia), due to the action of non-specific microorganisms. Specific microorganisms may sometimes invade an atelectatic focus. (Hogs develop the pneumonic type of hog cholera in this way.)

The **treatment** of acquired atelectasis depends upon the cause. In congenital atelectasis one must attempt to stimulate respiration by



massage of the heart (compression of the cardiac regions from both sides about fifteen to twenty times per minute), by artificial respiration (movements of the bent front leg upward and downward and then backward toward the region of the flank), the removal of mucus from the buccal cavity, rhythmical pulling out of the tongue, friction of the skin, instillations of vinegar into the nasal cavity, titillation of the nasal mucosa with a straw (Moussu). Hygienic measures, particularly proper breathing and proper care, are important from the standpoint of prophylaxis.

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## 7. Enzootic Pneumonia of Young Animals.

(*Septic pneumonia of calves, lambs, kids, pigs, foals; enzootic bronchopneumonia of young animals, pleuropneumonia septica* [POELS], *pneumo-enteritis septica* [GALTIER]; *Ferkelsterbe, Ferkel Husten, Zement Husten, chronische Schweineseuche* [German]; *Bronchopneumonie infectieuse des veaux, des agneaux, des chevreaux* [French]; *pneumonite dei neonati* [Italian].)

Enzootic pneumonia of young animals is an infectious pneumonia, or pleuropneumonia, which occurs enzootically, and is usually catarrhal, not uncommonly catarrhal croupous, or purely croupous; it is caused by various bacteria, among them the bacillus bipolaris septicus and its varieties.

Though some of the forms of pneumonia here enumerated have already been described in Volume I. as special diseases, the collective consideration in one chapter of all contagious pneumonias occurring in young animals cannot be avoided, because only in this manner is it possible to do justice to the requirements of practice and also to consider those forms of contagious pneumonias which were not taken up in the other volume.

**Historical.** Reports on the occurrence of a contagious pneumonia in young animals are found since the middle of the last century; but they received more particular attention since 1890. In 1886 Poels described the disease in cattle in Holland and claimed as its cause a bacillus similar to the bacillus of swine plague. These findings have subsequently been confirmed by Jensen (1890), van den Maegdenbergh and Liénaux (1892). Perroncito, in 1885, described a pleuropneumonia of calves, occurring sometimes sporadically and at other times epizootically. He claimed as its cause an organism which he called micrococcus ambra-tus, a bacterium which is different from the bacillus bipolaris septicus. The exact etiologic position of the disease described by Perroncito has not yet been cleared up satisfactorily.

Aside from those named, the following have studied the etiology of contagious pneumonia in young animals: Semmer (1888); Bongratz (1892); Stroese & Heine (1898); Grips, Glage & Nieberle (1904); Schreiber (1906); Pfeifer (1906); Hartl & Reisinger (1907); Lignières (1907); Berger (1907); and J. Müller (1907).

**Occurrence.** Enzootic pneumonia of young animals occurs in all countries in which young animals are bred in large numbers and it has been observed particularly in Holland, Germany, Denmark, France, Belgium, England, Italy, Switzerland, Russia, Hungary and America. Most commonly affected are calves, lambs, young pigs, less commonly kids, and rarely foals. The animals are, as a rule, affected during the first weeks of their lives, but even up to the sixth month and later.

The disease has become of greater economic significance since it recurs annually again and again in spring and becomes more extensive as a barn enzootic, causing numerous deaths and retarding the development of those animals which survive.

Ströse & Heine demonstrated contagious pneumonia in 1 to 1½% of all young hogs slaughtered in the stockyards of Hanover (Simader claims, however, that these were cases of pulmonary atelectasis). Grips, Glage & Nieberle saw pneumonia in about 50,000 young hogs in Hamburg.

**Etiology.** There is no uniformity in the etiology of the disease. The bacillus bipolaris septicus (see Vol. I) or its varieties (bacillus vitulisepticus, ovisepticus, suisesepticus, equisepticus) are frequently the cause of the disease. Galtier's pneumobacillus septicus is probably identical with the former organisms.

**Virulency.** The experiments of Poels have shown that the inoculation of cultures of bacillus vitulisepticus into the lungs or the serous cavities of rabbits kills the animals within fifteen to thirty-five hours; calves within twenty to sixty-six hours. Intrapulmonary infection is followed by a serofibrinous pleurisy, occasionally accompanied by lobular pneumonia. Rabbits, guinea-pigs and mice die from septicemia after subcutaneous injection or feeding. Hogs, when artificially infected, sicken under symptoms similar to those of swine plague; other animals, occasionally even guinea-pigs (Jensen), develop suppuration at the place of injection. Dogs are resistant. The experiments of Galtier with his pneumobacillus septicus were positive in intrapulmonary, intratracheal, nasal, intraperitoneal, intraocular and subcutaneous injections in calves, lambs, kids and hogs. Semmer, however, did not succeed in spreading the disease to healthy calves by the nasal, intratracheal and intrapulmonary injection of pulmonary juice. Bacteria, which were not fully described, were cultivated from the affected lungs, but did not prove effective in Greve's case; some other animals could not be infected. Similar were the (negative) results of Hartl & Reisinger in inoculating small laboratory animals with a pleomorphic bacterium similar to the bacillus bipolaris. After subcutaneous injections of this bacillus domestic animals susceptible to the disease did not usually become affected, though rabbits sometimes succumbed to a pneumonia (Stöhr). The same observations have been made with reference to the bacillus suisesepticus (Smith, Kitt) or to the bacillus bovisesepticus (Marek).

Several investigators found other bacteria, aside from the bacillus bipolaris as the cause of enzootic pneumonia in young animals. Poels, J. Müller and Schreiber observed an enzootic pneumonia in calves and young pigs, caused by a bacterium of



the coli group. Poels & Berger described an enzootic pneumonia in calves and young pigs, caused by streptococcus pyogenes. Bacillus pyogenes may likewise occasionally produce enzootic bronchitis and bronchopneumonia in young pigs and calves (Grips, Glage & Nieberle, Olt, Poels, Berger). The bacillus bronchiolitidis vitali described by Kitt is probably identical with the bacillus pyogenes. Poels also ascribes the faculty of producing enzootic pneumonia in young pigs to a staphylococcus pyogenes and to a kind of streptothrix, which he has not described more fully. Finally Berger observed an enzootic pneumonia in calves which was produced by bacillus pyocyaneus.

The causal relation between enzootic pneumonia and varieties of the bacillus bipolaris septicus is now generally considered as being well established. Still, many authors do not believe that the other microorganisms mentioned are at all etiologic factors in the production of enzootic pneumonia and hold that they are present only secondarily, having invaded an already diseased lung. This view appears justified in those cases in which bacteriologic examinations were made only in an advanced stage. However, other bacteria than the bacillus bipolaris have been found in the absence of the latter in very recent cases (Poels, Berger, J. Müller, Kitt, Grips, Glage & Nieberle, Olt) and pneumonia has been produced with them in experimental animals by subcutaneous and by intratracheal injection, as for instance by J. Müller with a bacillus of the coli group and by Junack with a staphylococcus or a streptothrix organism.

Several kinds of bacteria appear to be effective in some cases. Frequently, however, the presence of bacteria different from the bacillus bipolaris simply represents a secondary infection, particularly when the disease has run a longer course. This furnishes the basis for further tissue changes which may so modify the original pathologic picture that it appears as if we were dealing with a new disease (Holth). A secondary infection of the lungs may occur also in other enzootic diseases (pyosepticemia, dysentery of sucklings, white scours, strangles) and then give rise to numerous cases of bronchopneumonia (secondary bronchopneumonia of sucklings).

**The Etiological Relation Between Enzootic Pneumonia of Young Animals of Various Species.** Some incontestable clinical observations furnish the proof that enzootic pneumonia may occasionally be transferred from the young of one species to that of another. Lambs were infected from hogs suffering from swine plague (Keleti, Pr. Vb.). Calves were infected from calves, kids or foals suffering from septic pneumonia (Perroncito, Sauer, Pr. Vb., 1905, ii, 17, Bass, Pfeil); Rühm & Schreiber likewise consider the spreading of swine plague to calves, as probable or of calf-pneumonia to young pigs. In connection with the positive inoculation experiments mentioned, and the biologic properties of bacillus bipolaris, these observations permit the conclusion that the different forms of pneumonia caused by it in calves, pigs, lambs and kids are intimately related or are produced by varieties of the same bacterium. The bacillus bipolaris appears to have adapted itself, in the majority



of cases, to a certain species, so that only the young of the same species are susceptible to contagion. If, however, its virulency becomes increased, or if certain environmental conditions become favorable to microorganisms, it may spread to young animals of another species, and exceptionally to adult animals of this kind. (Pr. Vb., 1905, ii, 17.) Young animals may also be infected from adults, as occurs in hemorrhagic septicemia of cattle, in swine plague and in hemorrhagic septicemia of sheep.

The same general rule might apply to pneumonia of the young caused by other microorganisms, but observations proving this are still lacking.

The etiology of enzootic pneumonia of sucking foals has not yet been investigated; but the occurrence of this disease appears to be very probably due to some of the bacteria mentioned above. (Kutzbach observed an enzootic bronchopneumonia similar to that of calves among foals which had had an attack of strangles a few weeks previously.)

**Natural infection** occurs by sucking an unclean udder, through contact with straw, by licking the walls, through ingestion of infected food, through inhalation of droplets of secretion, which are coughed up by sick animals, through the stump of the umbilical cord, through abrasions of the skin. Sick animals confined to a stable will spread the infection by expectorated bronchial secretion, by intestinal discharges which may contaminate the floor, the straw, the feed remnants, the crib, and which may remain virulent upon the infected objects for a long time. The disease has, therefore, a tendency to become stationary in localities which have once become infected, unless prophylactic measures are carried out. Intrauterine infection, as claimed by Galtier, has not been proved to occur. The importation of the disease into hitherto free herds is usually brought about by young or adult animals which have passed through the disease, yet have not fully recovered. The disease is not rarely spread to new localities by obviously sick animals or by the attendants. It is spread most easily by sick animals of the same species.

The disease may make its appearance among animals which were heretofore free from the infection without importation from outside. If this occurs it must be assumed that the causative microorganisms have been present as saprophytes and have for some reason acquired pathogenic properties or that the power of resistance of the sucklings has for some reason been diminished by external influences (breeding of thoroughbred stock).

External influences, such as cold, sojourn in cold, drafty, poorly ventilated stables, favor the spread of the disease or cause a more virulent course. Another predisposing cause is primary intestinal catarrh or diarrhea (Poels) and too much in-breeding.

**Pathogenesis.** Some of the pathogenic bacteria mentioned above, especially bacillus bipolaris, multiply, in certain cases, very rapidly in the blood of the infected animals, and thus produce septicemia. If the latter does not lead to death in a short



time some of the bacteria circulating in the blood become colonized in the bronchi and the pulmonary tissues and bronchitis and pneumonia develop. In the meantime, the bacteria may have disappeared entirely from the circulating blood and remain only in the organs where pathologic changes are established. Other bacteria, and also the bacillus bipolaris, may produce pneumonia without a preceding septicemia by gaining access to the lungs either with the inhaled air or through the blood-current. The localization of bacteria from the circulating blood or their dissemination with the lymphatic current may cause inflammation of the serous membranes of the thorax, particularly if the bacteria possess a higher degree of virulence.

Since diseased pulmonary tissue forms a good nutritive soil for various bacteria, saprophytic microorganisms living in the bronchi of otherwise healthy animals or in their neighborhood may multiply rapidly (secondary infection) and produce further changes.

**Anatomical Changes.** These vary according to the several microorganisms and their variable degree of virulence, also according to the power of resistance of the affected animals and to the duration of the disease.

Aside from those cases which run their course as a pure septicemia and which are not here considered, in its **acute form** the disease not uncommonly presents the picture of a septic pleuropneumonia. A serofibrinous exudate is found in one or in both pleural cavities, the pleura being covered to an extent of several millimeters with loose, juicy, fibrin membranes, which are lusterless and studded with punctiform hemorrhages. The lung is uniformly dense, void of air, friable in its anterior lower portion; frequently also to a greater extent, a reddish-gray cloudy fluid may be scraped off the dark red-brown or grayish-brown, finely granular cut surface. The interstitial connective tissue appears uniformly serously infiltrated and forms yellowish, gelatinous, reticulated stripes of varying width on a red-brown or more grayish-brown background (marbled). Sometimes one sees lymph vessels with coagulated lymph and a fibrinous exudate in the wider stripes (Liénaux). Of other changes occasionally met with may be mentioned gelatinous infiltration in the neighborhood of the larynx and the pharynx, hemorrhages into the tissue of the pericardium and peritoneum, fibrinous pericarditis, acute swelling of the lymph glands, acute gastro-intestinal catarrh, sometimes ulcers in the abomasum (Beresow), cloudy swelling of parenchymatous organs, also, according to Galtier, hemorrhages into, and fatty degeneration of, the muscles of the rump, the extremities and of the heart. The anatomical picture in sheep and goats is sometimes similar to that of septic pleuropneumonia; in young pigs, however, to that of typical swine plague (see Vol. I).

Very frequent findings in acute cases are bronchitis or bron-



chopneumonia with or without a simultaneous pleuritis or pericarditis. Individual lobules or occasionally whole lobes (especially the anterior and median lobes and the anterior angle of the cutaneous lobes) appear brown, purple or grayish-red, flesh-like, dense, non-crepitant when cut into. The interlobular connective tissue appears occasionally somewhat swollen; the bronchi contain mucoid, gelatinous yellowish masses of secretion. The bronchial and mediastinal glands are markedly swollen.

The **chronic form** presents a flabby pneumonic consolidation. Occasionally one finds in the affected pulmonary tissue purulent or caseous foci from pea to walnut size, in other cases dry, grayish yellow necrotic foci, sometimes also cavities as large as a fist. The pleurae present the picture of a chronic fibrous adhesive inflammation. The bronchial and mediastinal lymph glands often show considerable chronic swelling and exceptionally contain dry foci up to the size of a lentil.

Pneumonia assumes the form of bronchopneumonia suppurativa if caused by bacillus pyogenes (Olt, Holth) or bacteria of the coli group (J. Müller). It is then characterized by a light yellow suppurative condition of the small bronchi, in the middle of the hepatized or only atelectatic lobules. A gradual purulent liquifaction of lobules, inspissation of pus and adhesive pleurisy radiating from the bronchioles takes place in the further course of the disease.

Kitt described a caseous bronchopneumonia in a young steer which was very similar to pulmonary tuberculosis, but differed from it in that there were no caseous foci in the connective tissue between the white yellowish caseous looking lobules; calcareous changes and cavity formation was likewise absent. The exudate in the bronchi contained exclusively the bacillus bronchionitis vituli (see page 135).

**Symptoms.** The duration of the period of incubation varies a good deal according to the type and virulence of the pathogenic bacteria, and it can be determined only in cases with an acute course.

According to Schreiber septic pneumonia occasionally appears on the second day of life; in other cases on the ninth to seventeenth day after infection. In contagious catarrhal pneumonia of young pigs Grave observed a period of incubation of ten to fourteen, rarely up to twenty-four days.

The clinical picture itself varies a good deal. One can distinguish, generally speaking, an acute and a chronic form of the disease.

The **acute form** is usually observed in very young animals a few weeks old at the utmost; it is initiated by a decrease in the lively behavior and in appetite, and by a febrile elevation of temperature up to  $41^{\circ}$  to  $42^{\circ}$  C. The respiration is accelerated from the start and becomes more and more frequent, so that after a few days the number of respiratory movements may be 40-60 even 100 per minute. At the same time the respiration becomes forced, the animals spread their front legs, stretch their heads and neck; calves and foals also dilate their nostrils. Cough occurs soon after the onset of the dis-



ease; at first it comes on during exercise; later on also during rest and it becomes more frequent and more tormenting. Mucopurulent nasal discharge occurs in some cases.

Percussion and auscultation of the chest reveal the same changes as are usually found in bronchopneumonia (see page 129). In some cases there are found in addition the signs of acute fibrinous or serous pleurisy or pericarditis. Exceptionally the disease may present itself exclusively under the clinical picture of acute pleurisy or pericarditis (Immelmann).

The physical weakness increases rapidly during the course of the disease; the animals become unable to stand up and they succumb between the second, fourth, sixth day of the disease; often profuse and fetid diarrhea has set in. If, as occurs rarely, the animal remains alive the symptoms of the chronic form of the disease are developed.

The **chronic form** is seen in somewhat older animals or in somewhat milder outbreaks; cough being the first obvious symptom of this form of the disease. It is at first infrequent and comes on only on rising or lying down or during exercise. Later on it becomes more frequent and weaker. In the meantime, the respiration becomes more frequent and more or less forced. Râles and whizzing sounds, also purring are heard over the chest and in addition often the changes on percussion and auscultation as in the acute form. Fever is often present; only of moderate degree. The general condition and the appetite may remain undisturbed, particularly in hogs; however, the appetite is, as a rule, diminished and the sensorium more or less depressed.

Gradual emaciation becomes manifest in the further course. In this manner a chronic marasmus is established, which may terminate fatally in three to six weeks or only after one to two months. In the milder cases the condition gradually improves, the cough becomes less frequent, the nutrition better, and all symptoms entirely disappear, often however, only after several months.

In some cases, apparently in those due primarily or secondarily to the bacillus pyogenes, itching and scabby eczemata, appear on the neck along the back, on the root of the tail, also swellings and abscesses in various parts of the body, which are, according to Schimmelpennig, of unfavorable prognostic significance.

**Diagnosis.** The exclusive affection of young animals and enzootic occurrence of the disease are generally sufficient to lead to a correct diagnosis, and there is frequently besides the additional chance to make a post-mortem examination on one of the earliest fatal cases. The exclusive affection of calves, lambs or kids distinguishes the disease from pleuropneumonia in cattle and from hemorrhagic septicemia of sheep and goats. The same is true of the differential diagnosis between contagious pneumonia of young pigs and typical swine plague which is caused by bacillus suissepticus alone, while in hog cholera the clinical and anatomic pictures are usually charac-



teristic. A favorable course also speaks against swine plague. In doubtful cases the epizootical factors have to be considered. Contagious pleuropneumonia can usually be easily distinguished from pneumonia of calves according to the principles laid down in Volume I. An enzootic occurrence permits the exclusion of sporadic bronchopneumonia. Lungworm disease is usually seen in somewhat older animals after pasturing. The detection of worms, their embryos and ova in the coughed up sputum, or on post-mortem examination in the bronchi, establishes the diagnosis beyond doubt. Atelectasis of the lung must also be considered in the anatomical diagnosis (see page 131). The fact that calves and lambs hold their bodies stiff might lead to a confusion with tetanus; a more exact examination will, however, exclude such an error.

The detection of the causative microorganism should be attempted in all cases by post-mortem examination and by bacteriologic tests of recent cases.

**Prognosis.** It is usually quite unfavorable; however, considerable variations occur in various enzootics as to the morbidity and mortality, according to age, species and environmental conditions of the animals. Most of the animals which are affected during the first weeks of life perish and even those that remain alive are stunted in their development, and their raising does not pay. A complication of dysentery is particularly unfavorable. The disease very often terminates favorably in young pigs over one month old (Greve, authors' own observation), and also not uncommonly in other animals; the recovery may, however, be apparent only and extensive indurations may persist in the lungs.

**Treatment.** Medicinal treatment is not at all promising and its carrying out meets with great difficulties. Dietetic and hygienic regulations may, on the contrary, influence the course of the disease favorably (see page 120). Serum treatment may be indicated in those forms of pneumonia which are caused by the bacillus bipolaris, by bacteria of the colon group and by streptococci.

**Prophylaxis.** Prophylactic measures carried out energetically permit the successful struggle against enzootic pneumonia of young animals; hence our main efforts must be spent in this direction. To prevent the importation of the disease, it is advisable not to introduce too young animals for breeding purposes and to subject newly bought stock to an isolation of two weeks and then to keep them together with other young animals only provided they are healthy and have not shown any suspicious symptoms (cough!). The stables must be kept clean and must be properly ventilated; during favorable weather the animals must be kept in the open and inbreeding must be avoided. The floors of stables and pig pens should be warm; cement or stone floors are not desirable and if they

are present, they should be covered with lumber, which can easily be removed and cleaned.

If, in spite of precautions, the disease has made its appearance, healthy animals should be separated from the sick and suspicious ones. The removal of the healthy animals and their mothers to another stable or another farm, etc., where there are no young ones is very desirable. Those animals which are to be transferred should be cleaned, especially on their extremities. The isolated animals should have separate attendants. If the disease has made its appearance to a limited extent, it is best to slaughter the sick and suspicious animals, otherwise they may be kept alive and isolated until they either recover or until their slaughter becomes necessary. Recovered animals should only be used for breeding purposes with every possible precaution, since they may become instrumental in spreading the disease.

Stables, pens and runs must be disinfected thoroughly after the removal and burning of loose woodwork. Manure must be made innocuous by the use of chloride of lime. Stables for breeding animals, which have been visited repeatedly by the disease, must be disinfected before the young ones are born (the procedure after parturition has been indicated in Vol. I).

These measures usually make it possible to stop the spread of the disease and to prevent its occurrence.

**Protective Inoculation.** Protective inoculation against pneumonia of lambs has recently been practiced with good results (Proske, Goldmann, Beckhard, Becker) with a polyvalent immune serum of Ostertag & Wassermann (Pr. Vb.) or with septizidin or with septizidin B. (Goldberger, Evers, Schreiber). Dysentery serum has been used in other cases. Streptococcus serum is indicated in streptococcus infections. It appears advisable to vaccinate animals shortly after birth and to repeat the vaccination after a few weeks.

It is impossible at present to make a definite statement on the prophylactic efficiency of protective inoculation; it must, however, be stated that unfavorable results have also been reported. This may, of course, be explained by the fact that enzootic pneumonia of young animals is caused by a variety of bacteria, and that the virulency of the bacillus bipolaris is subject to a great deal of variability.

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## 8. Mycotic Pneumonia. Pneumonomycosis.

(*Pneumonomycosis aspergillina*.)

**Etiology.** Moulds in the air passages are found most frequently in places where the temperature is somewhat lower and where fungi can remain permanently in contact with the air. They also form larger colonies in the presence of more abundant masses of secretion or decomposition products. One finds colonies of moulds in bronchiectasias, pulmonary cavities, exceptionally in bronchial catarrh. In all of these cases, however, moulds live in the interior of the air passages only as saprophytes without attacking the living tissues and without producing pathologic changes in them (secondary mycosis).

Only rarely can disease of the air passages be referred directly to a pathogenic effect of moulds (primary or true mycosis). This appears to be the case after the inhalation of very great amounts of moulds. Moulds may display a pathogenic effect preferably in weakened individuals and in the presence of a catarrhal affection of the lungs. Schultz produced the disease artificially in birds by inhalation. According to Fölger, mould spores may also get into the lungs by embolic transport.

Most common of the pathogenic moulds are species of *aspergillus*, especially *aspergillus fumigatus* and *aspergillus nigrescens*. The former still grows well at a temperature of 37°-40° C.; the latter at 37° C.; hence *aspergillus fumigatus* is considerably more dangerous (Schultz, Lucet). *Aspergillus glaucus* does not grow at body temperature and only forms larger colonies perhaps in the larger air passages, but it cannot penetrate into the tissues. Some species of *mucor* grow at 40° C. and also in the interior of animal tissues.

The species of *aspergillus* form a colorless mycelium, from which arise straight, non-branching hyphae; these end free in a globular swelling, the columella, which carries numerous radially arranged sterigmata, from which a single row of conidia is formed by constriction, which, in combination with the sterigmata and the columella form the fruit-headlet (Fig. 19). The following species are pathogenic:

*Aspergillus fumigatus* forms at the beginning bluish-green, later on ashy-gray colonies. The diameter of the semispherical or club-shaped columella is 8 to 20  $\mu$ ; of the conidia 2 to 3  $\mu$ . The sterigmata rise more or less upward.

*Aspergillus nigrescens* (asp. niger.) columella spherical and carries pure radially arranged sterigmata, with blackish conidia. The mycelium therefore looks chocolate colored.

*Aspergillus glaucus*; the hypha  $\frac{1}{2}$  to 1 mm. long possesses a club-shaped columella with radially arranged sterigmata. The segmented conidia are grayish-green, the mycelium is of the same color (Fig. 19).

Characteristic of *Penicillia* (brush-moulds) is an umbelliferous arrangement of the conidia bearers, which terminate in bottle-shaped cells, each one of which carries a chain of conidia (Fig. 20). Pathogenic penicillia have so far not been found in domestic animals.

The fruit bearer in *Mucorinae* terminates in a spherical brown or black sporangium containing the spores. After rupture of the smooth membrane these escape into the outside world (Fig. 21). *Mucor racemosus* and *mucor conoideus* have heretofore been found in the air passages.

**Natural infection** takes place on living in musty, moist-warm, ill-ventilated spaces and in consequence of the ingestion of mouldy feed. Animals which get but little into the open air and which are feeble or weak appear to be predisposed. The disease is, therefore, most common among domestic fowls, since they are comparatively frequently kept in mouldy, moist, poorly ventilated places. Primarily affected are pigeons, chickens, ducks, geese, sometimes also other birds, and even cage birds (John saw pneumonocystis in nineteen flamingoes of a zoological garden; other authors have seen the disease in parrots). Mycotic pneumonia has more rarely been seen in mammals; Schultz, Rivolta, Martin, Bollinger, Lucet, Peck saw the disease in horses; Röckl, Piana, Bournay, Ravenel, in cows; Hartenstein, in calves (in eleven calves in Hungary); Hellews, in sheep in enzootic extent; Mazzantini, in a lamb; Serrurier & Rousseau, in a stag; and Rivolta, in a dog.

Fig. 19. *Aspergillus glaucus*.Fig. 20. *Penicillium glaucum*.

### Anatomical Changes.

Dirty yellow or greenish, mouldy looking, dry deposits are formed on the mucosa of the bronchi and on the internal surface of the air cells in birds;



under these deposits the tissues are sometimes ulcerated and they may completely obstruct the bronchial lumen. In the



Fig. 21. *Mucor mucedo*.

pulmonary tissue catarrhal foci are sometimes found; sometimes disintegrated tissue composed of purulent caseous or more grumous nodules and surrounded by a connective tissue capsule or by an infiltrated, occasionally hemorrhagic, focus, which is at other times imbedded in a hepaticized tissue (Martin). On microscopic examination the deposits and the foci show the moulds. This dif-

ferentiates definitely mycotic foci from caseous tuberculous foci or glanders nodules, to which they may be very similar. Hepatization of neighboring nodules and of the interstitial connective tissue may in cattle simulate the picture of pulmonary tuberculosis (Röckl). In horses the changes may be similar to pulmonary gangrene (Pech).

**Symptoms.** In its symptoms and course the disease is frequently similar to catarrhal pneumonia, and the clinical picture is therefore initiated by the symptoms of a bronchial catarrh.

In birds the respiration becomes rattling, gasping and more and more difficult. The appetite is poor, but the thirst is much increased. The animals do not like to move; their wings droop and they sit long in one place with ruffled feathers and somnolent. The buccal mucosa sometimes shows yellowish or greenish deposits (Potain). The emaciation progresses more and more; diarrhea comes on and the completely exhausted animals die after a period of four to eight weeks' duration.

Symptoms pointing to a chronic pulmonary disease are observed in **mammals** and may exist for weeks and months; the animal suffers from increasing respiratory difficulties, they become emaciated to skeletons and completely exhausted. More rarely the disease takes a rapid course and then leads to more acute symptoms, eventually with a hemorrhagic discharge from the nose and to hematuria (Thary & Lucet).



**Diagnosis.** The disease can be diagnosticated with certainty only after the microscopic detection of the moulds in the secretions of the air passages, after the history and the development of the disease have led to a suspicion of pneumomycosis. Microscopic findings are insufficient, because similar moulds may be found in the air passages, particularly of mammals, as harmless saprophytes.

**Treatment and Prophylaxis.** To prevent the further multiplication of the moulds, inhalations with disinfecting solutions, or intratracheal injections (see pages 42, 43) may be tried. Their success appears very doubtful, because they do not reach the moulds which are located in the alveoli. One might try the methodical administration of iodide of potash.

The most important measures are prophylactic. The animal houses, especially those of fowls, must be kept clean and dry, and the feeding of mouldy material must be avoided. To be recommended are good ventilation, drying out and cleaning of the places, washing of the walls and wooden portions with hot water or a disinfecting solution.

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## 9. Purulent Pneumonia. *Pneumonia suppurativa.*

(*Abcess of the lung. Pneumonia embolica. Abscessus pulmonum.*)

**Etiology.** A frequent cause of purulent pneumonia is the lodgment of infected emboli, especially during the course of septicopyemia of sucklings. Other sources of infected emboli may be: purulent metritis after parturition, abscesses of strangles, ulcerous endocarditis, purulent or gangrenous wounds in various places of the body (pressure gangrene, gangrenous inflammation of the feet), fistulæ formed after venesection or castration, etc. Foreign bodies, and with them pyogenic bacteria, more rarely get into the pulmonary tissue. Such foreign bodies may enter the lungs from the air passages after injury to the thoracic wall from the stomach.

Suppuration of pulmonary tissue most rarely follows croupous or catarrhal pneumonia (concerning catarrhal purulent pneumonia caused by the bacillus pyogenes in cattle and swine, see page 126).

In some forms of catarrhal pneumonia or independently from it the bacillus pseudotuberculosis of Preisz forms greenish yellow, soft caseous foci (see Vol. I), and more rarely the bacillus necrophorus causes in catarrhal or croupous pneumonia more dry and caseous foci.

**Anatomical Changes.** In acute cases cavities are found in the lungs filled with pus and tissue detritus; their internal

surface is uneven and villous, the surrounding pulmonary tissue is tough and void of air in consequence of cellular infiltration. On the other hand, whole pulmonary lobes may be infiltrated showing on the cut surface grayish-yellow points of the size of a pin head. Older abscesses have their own connective tissue walls and well established cases show inspissated pus or dry caseous masses in their interior. The pleura shows evidences of a seropurulent or purulent inflammation whenever the abscesses are situated superficially.

**Symptoms.** The symptoms of the disease vary very much from case to case. If suppuration has followed upon croupous

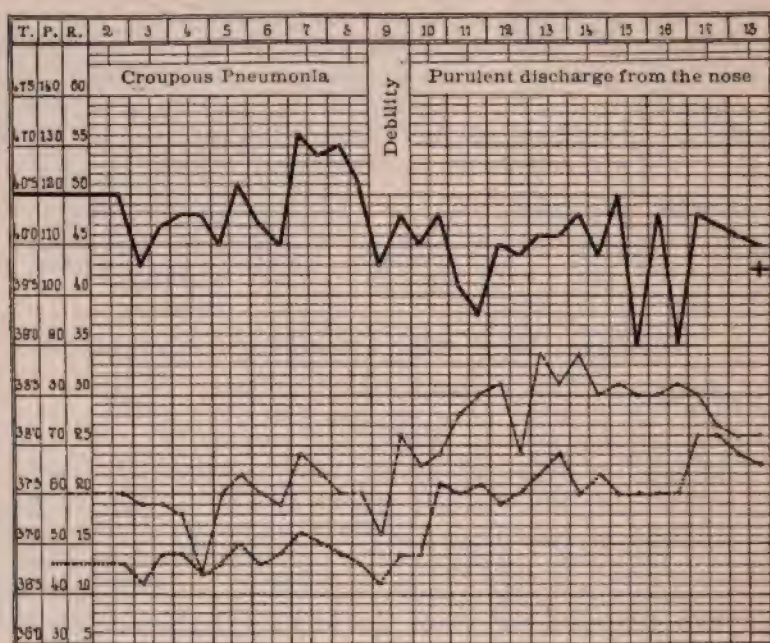


Fig. 22. Fever curve in purulent pneumonia in a horse.

or catarrhal pneumonia, the liquefaction of the exudate is retarded, an elevation of temperature again occurs and the grave symptoms of acute pneumonia again stand in the foreground of the clinical picture. Embolic purulent pneumonia develops rapidly and leads in a short time to severe symptoms.

The symptoms point to grave acute pneumonia with high fever (Fig. 22) and respiratory disturbances; later on the signs of a septic infection are present. Percussion either shows no deviation from the normal or there may be more or less dullness, or tympanitic sounds in circumscribed places; eventually also metallic sounds. One hears râles, sometimes also bronchial

or amphoric breathing. The picture of pulmonary gangrene is frequently developed (q. v.).

The **diagnosis** can be established beyond doubt only if an abundant, purely purulent nasal discharge is present and if we find in it on microscopical examination elastic fibers (Fig. 23), fat crystals and possibly also necrotic pulmonary tissue.

**Course.** The disease terminates, as a rule, fatally within a very short time; occasionally after only one to two weeks. The immediate cause of death is usually sepsis, also sero-fibrinous, purulent, sometimes gangrenous, pleurisy. Recovery is very rare, but it occurs occasionally. Abscesses may become encapsulated or they may break into a bronchus and finally heal, as was seen several times by John. It is quite difficult to diagnosticate such slowly healing abscesses on the living animal.

**Treatment** is usually unsuccessful. In practice stimulating drugs may be employed, also disinfection of the air passages and pneumotomy in case of superficial abscesses (Pansini, O. M., 1907, 289).

#### 10. Pulmonary Gangrene. *Gangraena Pulmonum*.

(*Gangrenous pneumonia, foreign body pneumonia, pneumonia gangrænosa.*)

By gangrene of the lungs is meant the putrid decomposition, by saprophytic bacteria, of pulmonary tissue which has previously become necrotic.

**Etiology.** Necrosis of pulmonary tissue is usually brought about by inflammatory processes, more rarely by injury to the lungs. Putrid bronchial catarrh frequently plays a rôle and it usually develops after aspiration (aspiration pneumonia). It occurs most frequently in horses, not as frequently in cattle, rarely in other domestic animals, and is then usually associated with disturbances of deglutition. As the cause of the latter there is usually found acute inflammation of the pharyngeal mucosa or of the adjoining organs. Disturbances of deglutition may be caused mechanically by abscesses in the pharyngeal wall or in the retropharyngeal lymph glands, by tumors, foreign bodies in the pharyngeal wall or in the air sac, paralysis of the pharynx, convulsions of the pharyngeal muscles (in tetanus or meningitis). Any disease associated with grave disturbances of consciousness may likewise cause aspiration. Aspiration occurring in paralysis of the pneumogastric nerve (as, for instance, in chronic lead poisoning—Thomassen) and so-called



vagus pneumonia, following it, is dependent upon anesthesia of the larynx and the air passages, and upon paralysis of the muscles of the larynx, which makes the removal of the foreign bodies that might get into the larynx impossible. Nutritive disturbances of the pulmonary tissue may, according to Bettini, likewise take part in it. The sudden entrance of larger amounts of fluid into the pharynx may likewise give rise to aspiration as occurs in the breaking of a peri- or retropharyngeal abscess, in obstruction of the esophagus, during vomiting, in sudden falling down of cattle, in malignant foot-and-mouth disease; also in consequence of the protrusion of the contents of the paunch. In protracted labor a fetus sometimes aspirates amniotic fluid that is already contaminated. Foreign bodies, such as ears of grain, pieces of wood, hairs, are sometimes aspirated. In the pleural cavity of a dog, found dead from pulmonary gangrene, Mori once found six ascaris worms. He believed that they had been aspirated in vomiting and had gotten into the lungs and from there into the pleural cavity.

Artificial feeding or the administration of liquid medicines may become dangerous in more serious disturbances of deglutition. Pneumonia caused by aspirated medicines may occur without the simultaneous existence of disturbances of deglutition (drenching pneumonia, pneumonia medicamentaria), if the medicines are administered awkwardly, if they are given at one time in too large doses, if the tongue is pulled out, or the nares are closed, if the head is held too high, if the drenching is continued in spite of cough or squealing (in hogs), if the fluid is poured into the nose, if the medicine cannot be swallowed easily, or if it produces attacks of cough.

Various, including putrefactive, bacteria, which in such cases have gained access with the aspirated substance into the bronchi, then multiply and produce first a severe inflammation in the bronchial wall, and the inflammatory process may be followed by necrosis and putrefaction. The inflammation spreads through the bronchial wall and also along it into the pulmonary parenchyma; here it produces catarrhal and hemorrhagic inflammation and soon also necrosis and putrefaction.

Of the various types of pneumonia, croupous pneumonia of horses and swine, preferably and comparatively frequently passes into pulmonary gangrene; catarrhal pneumonia leads less frequently to this process.

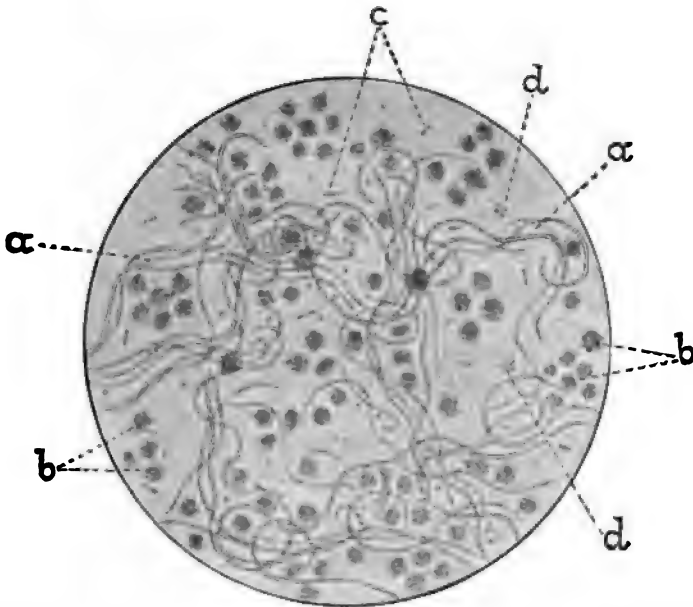
The contents of abscesses and caseous foci (tuberculosis, echinococcus, etc.) may become putrid; bacteria which have entered with the inhaled air are then responsible for this process. Infected emboli may carry the organisms (in Nielsen's case bacillus necrophorus) from other gangrenous or ulcerating organs into the lungs.

Injury to the lung may directly become the cause of pulmonary gangrene if putrefactive bacteria gain entrance at

once or later on, or if portions of lung have been crushed. Injury to the lung may be brought about by external violence (wounds, fractured ribs) or, particularly in cattle, by foreign bodies which enter the lungs from the stomach or pericardial sac.

Foreign bodies do not always cause gangrene but sometimes only an interstitial pneumonia, as is demonstrated not so very rarely by findings on slaughtered animals. The foreign body may even be coughed up (Lisizin).

**Anatomical Changes.** Gangrenous foci, usually quite numerous, are found in the lungs, especially in their anterior and inferior portions. These foci appear dirty-brown red or dirty-



**Fig- 23.** Nasal discharge in pulmonary gangrene of the horse. *a*, elastic fibers from pulmonary tissue; *b*, pus corpuscles; *c*, bacilli; *d*, cocci.

yellowish brown; they consist, either at the periphery, or throughout their entire extent, of a mushy mass of very disagreeable, sweetish, foul smell. Liquefied or soft masses, which have a similar smell and are likewise discolored, are found also in the bronchi, the mucosa of which is dirty red or slate-gray in color. In the neighborhood of the gangrenous foci and between them, the pulmonary tissue shows the picture of a catarrhal or croupous pneumonia. The internal surface of the cavities that are formed is ragged, eaten out, and is covered with a friable, smeary, stinking, mushy mass. In the rarer more chronic cases one finds in the neighborhood of the cavities a yellowish, or yellowish-gray, purulent, infiltrated zone or a cicatricial gray shell of connective tissue.



Foci situated near the pleura frequently lead to a purulent or putrid pleurisy, or pneumothorax develops after the focus breaks through. The internal organs present the signs usually found in grave general infection, such as parenchymatous and fatty degeneration, occasionally also hemorrhages and metastatic foci.

**Symptoms.** The earliest symptom of pulmonary gangrene is usually a peculiar, sweetish, foul, very disagreeable smell of the exhaled air. Stinking gases are developed in consequence of the putrefactive processes; these escape with the exhaled air and are at first noticeable only in the immediate neighborhood of the nose of the animals; later on they pervade the air of the neighborhood and can be smelled at once upon entering the place where the patient is kept. This smell may, however, be absent in pulmonary gangrene, if the focus has remained closed and has no communication with the outside world through the air passages.

As soon as gangrenous foci have broken into bronchi, there appears a dirty-grayish red, eventually brown-red or greenish tenacious nasal discharge, which becomes very abundant after coughing or after lowering of the head and which always disseminates the disgusting smell. Microscopically, the nasal discharge (Fig. 23) shows granular tissue, detritus, fat droplets, needle-shaped fat crystals, brown or black masses of pigment, sometimes pus corpuscles, red blood corpuscles, numerous varieties of bacteria, and elastic fibers, occasionally in alveolar arrangement. (Elastic fibers can easily be demonstrated after boiling the exudate in 10% solution of caustic potash, followed by centrifuging.)

Corresponding to the extent of the inflammatory process, the respiration is difficult and accelerated; this becomes particularly marked after a general septic infection of the organism has taken place.

The percussion sounds remain unchanged if central portions of the lung are affected exclusively. Frequently, however, we hear dullness, and occasionally also tympanitic sounds in the anterior and inferior pulmonary portions. The formation of cavities may sometimes be diagnosed if elastic tympanic, or metallic sound, or cracked pot sounds are found in a circumscribed place of the thorax, associated with metallic râles and bronchial breathing; these correspond in pitch with the tympanitic percussion sound. Splashing and similar noises are also characteristic of the existence of pulmonary cavities.

Pulmonary gangrene is almost always accompanied by fever. Its intensity varies from case to case, but it usually reaches above 40° C. and shows a remittent type (see Fig. 24); an insignificant fever is often seen at first for several days in aspiration pneumonia. The pulse becomes accelerated, small,



and soon thready. Chills and sweats come on from time to time. There is great prostration and dullness of the sensorium, the appetite is suppressed and profuse diarrhea not uncommonly comes on toward the end of the disease.

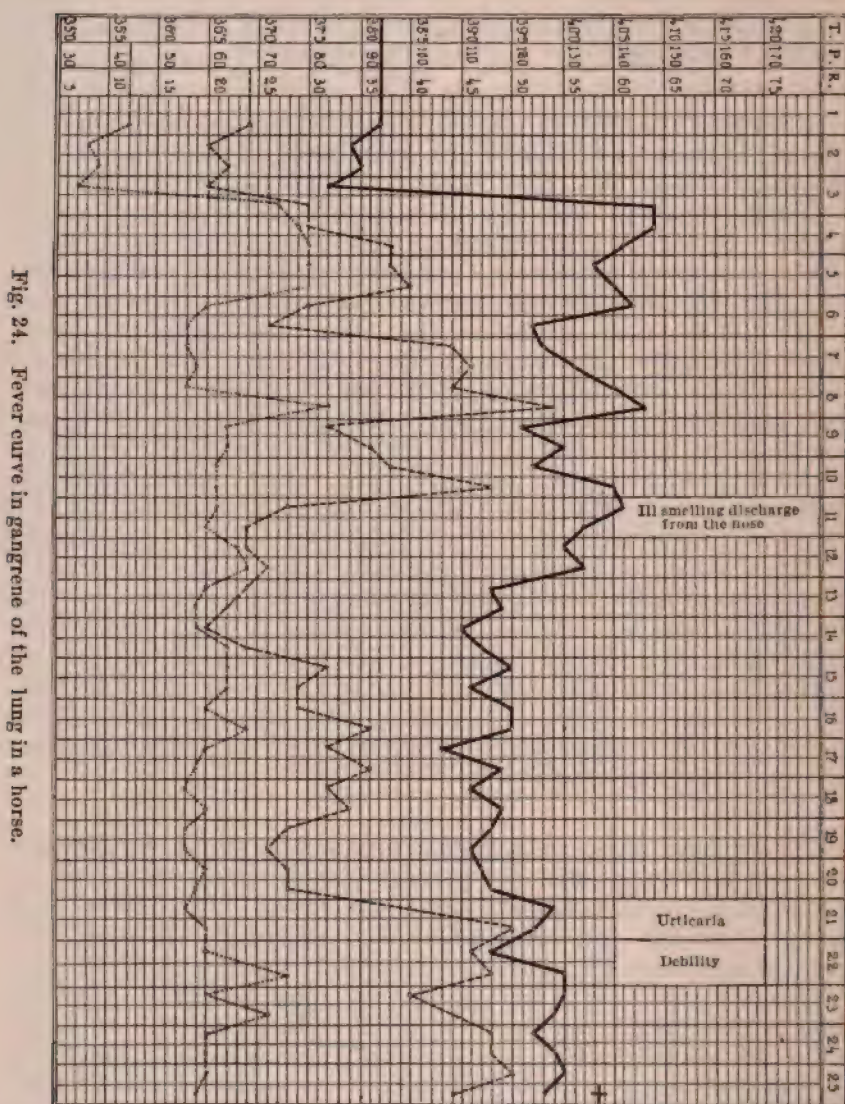
Pleurisy frequently develops during the course of the disease; pneumothorax only exceptionally. Pulmonary hemorrhage from the rupture of a blood vessel by coughing or by ulcerative processes of the vessel walls is rare.

**Course.** If pulmonary gangrene follows upon some form of pneumonia, it is preceded by the symptoms of this disease. If gangrene occurs in the course of croupous pneumonia, it usually occurs toward the terminal stage of hepatization; the non-appearance of improvement or resolution, or a new elevation of the temperature which had been falling, then point to an aggravation of the condition. It is also possible that the symptoms of pulmonary gangrene become manifest only after the beginning of the stage of resolution; a putrid inflammation of parts situated more centrally may at first not become manifest and may only be suspected after the fetid odor of the exhaled air and of the nasal discharge have been observed. In all cases, however, after once being established, the gangrene progresses rapidly to a fatal termination, and the animals die between the second and fourth, or in any event before the end of the eighth day. Pulmonary gangrene arising after other diseases than croupous pneumonia, usually takes a similar course, except that sometimes weeks may elapse before a more considerable portion of the lung has become affected. The symptoms are then at first mild and only gradually increase in severity. A gangrenous pulmonary focus may, in exceptional cases, become encapsulated and recovery take place.

**Diagnosis.** The only pathognomonic sign of pulmonary gangrene is the presence of shreds of pulmonary tissue or of elastic fibers in alveolar arrangement found in the nasal discharge or in the sputum expelled by coughing. Without the detection of these elements, the diagnosis depends upon the peculiar smell and upon the detection of signs pointing to cavity formation in the lung. In other cases, a diagnosis can be made only with some degree of probability; it becomes, however, fairly certain as soon as the peculiar disagreeable smell becomes manifest in the course of acute pneumonia, or if the symptoms of pneumonia come on after a disease which points to the aspiration of particles of food, masses of secretion, etc. Signs of sepsis are also of importance and always accompany pulmonary gangrene of any extent.

The disease might be confounded most easily with putrid bronchial catarrh; if primary, this does not lead to any consid-

erable elevation of temperature, or to pulmonary consolidation; it is, however, always present as a concomitant factor in pulmonary gangrene. The differential diagnosis becomes particularly difficult if putrid bronchial catarrh is present in connection with bronchiectasis; the constancy of abnormal percussion



sounds and rather insignificant disturbances of the general condition speak in favor of bronchiectasis. In gangrenous inflammation of the upper respiratory passages, especially of the pharynx, the nose and its accessory cavities, one finds, aside from the putrid smell of the exhaled air, other symptoms point-

**ing** to the organ affected in particular, and the grave symptoms of affection of the lungs are absent; one can sometimes ascertain that only the air exhaled from one nostril has a putrid smell.

**Prognosis.** While a gangrenous process in a circumscribed territory of the lung may, in rare cases, come to a standstill, be encapsulated, and end in permanent recovery (if occurring at all, most commonly in cattle and swine), the process is, as a rule, progressive in character; sepsis develops and a fatal termination occurs. The prognosis is therefore, in general, unfavorable, and the more so when gangrene is established in a previously inflamed lung or as a complication in a grave general infection.

**Treatment.** One might try inhalations of finely atomized disinfectant solutions, or intratracheal injections, the latter after a preliminary tracheotomy; it may exert a favorable influence upon the upper respiratory passages and may diminish the fetid smell (see page 61). A circumscribed gangrenous focus may possibly be removed by pneumotomy (Pansini, Ö. M., 1907, 289).

Further therapeutic measures consist in strengthening and nourishing the patient, in the regulation of the cardiac action, combating hyperpyrexia, and ameliorating cough and diarrhea.

As a matter of prophylaxis, cases where it is advisable to guard against the development of gangrene, where the exhaled air becomes fetid, should receive inhalation-sprays, or still better, intratracheal injections of some disinfectant solution (see page 61). One must always guard against aspiration in the administration of medicine, and therefore one should entirely avoid drenching if disturbances of deglutition exist.

## 11. Chronic Interstitial Pneumonia. *Pneumonia interstitialis chronica.*

(*Pneumonia indurativa, Sclerosis pulmonum, Cirrhosis pulmonum.*)

**Etiology.** Chronic interstitial pneumonia occurs very rarely in horses and cattle as a primary disease. Among these must be mentioned particularly what has been described by Dieckerhoff, and later by Bang, as lardaceous pneumonia in older horses; it appears to develop after an infection. (Bang expressly states that the disease has nothing to do with tuberculosis, but Rabe claims to have found tubercle bacilli in a case of this kind.) A disease, in some respects quite similar to that mentioned, has been described by Grüter, a pneumonia developed in connection with bronchitis scleroticans in horses. A



further cause of chronic pneumonia may be the inhalation of dustlike bodies (fragments of plants, silicon-, lime- or coal-dust), which are absorbed from the bronchi into the interstitial pulmonary tissue, and there produce a usually circumscribed, occasionally, however, a more extensive, chronic inflammatory process. (Pneumoconiosis.) Larger foreign bodies which have penetrated into the lungs from the air passages or from the fore-stomach may exceptionally produce chronic interstitial pneumonia. Schmidt observed a traumatic inflammation of the stomach, diaphragm and lungs in a horse with bronchiectasis.

Vansteenberghe & Grisez claim, on the basis of their animal experiments, that pulmonary anthracosis is not caused by the inhalation of dust-like foreign bodies, but by the absorption of dust-like bodies, which are swallowed and only secondarily brought from the intestines into the pulmonary vessels. Numerous experiments which were stimulated by the above statement have, however, proved that the dust-like foreign bodies in natural affection do get into the lungs by inhalation; anthracosis of intestinal origin is observed only exceptionally and then it does not reach a high degree (Lüttschwager).

As a rule, the disease is a secondary affection, seen particularly after bronchial catarrh. When existing for a long time, this leads to the new formation of connective tissue in the bronchial wall and finally spreads to the neighboring interlobular connective tissue. Purulent catarrh of the bronchi, giving rise to chronic pneumonia, occurs frequently in cattle, hogs and sheep. Polyadenomatous proliferations in sheep (Prösch) and cats (Ball) may have a similar origin, but they are rather to be looked upon as true tumors (q. v.). The process arises not uncommonly from atelectatic foci (see page 131). In protracted cases of acute pneumonia a progressing inflammation in the connective tissue is also seen, surrounding the affected lobules. This termination is observed particularly after catarrhal pneumonia, more rarely after croupous pneumonia, where, if seen at all, it occurs usually in old debilitated animals. An inflammatory new formation of connective tissue also occurs in the neighborhood of encapsulated abscesses, putrid or caseous foci, parasites, tumors, etc. Chronic pneumonia also occurs in the course of chronic pleuritis. Certain chronic infectious diseases also always finally lead, in their localization in the lungs, to chronic connective tissue inflammation.

**Anatomical Changes.** The affected pulmonary portion appears firmer, tougher, more tenacious, it contains less air, the cut surface shows a few cicatrices or cicatrixlike stripes between the air-containing lobules, or a whole portion of lung may be changed into an almost homogenous, firm, airless tissue, in which one can see only here and there an air-containing lobule. The shrunken portions of lung sometimes contain caseous foci and abscesses.

In lardaceous pneumonia of horses the upper portions of



the lungs appear uniformly grayish-white or yellowish-gray and lardaceous. They contain little air and are, consequently, firm. The lower and inferior portions usually show only a few nodules and are otherwise similar. Microscopically, we find proliferation of alveolar epithelia, much new formation of connective tissue, compression of the alveoli; the histologic picture has some similarity to that of carcinoma. There is, on the other hand, some similarity between lardaceous pneumonia of horses and multiple adenoma of sheep (see tumors of the lungs), and also between focal chronic pneumonia following bronchitis scleroticans of the horse. However, in this form of chronic pneumonia, inflammatory new formation of tissue is found, particularly in the peribronchial tissue, along the blood vessels and in the interalveolar tissue (bronchopneumonia fibroplastica, Grüter). In this latter disease lardaceous-looking foci are also generally seen which, however, grow at most up to the size of a fist.

In pneumoconiosis one sees in the lungs either fibrous-calcareous nodules or streaky cicatricial bands, or on the contrary, the picture of a fibrous bronchopneumonia; after coal-dust inhalation slate-black spots are also observed. The anatomical picture usually includes the signs of chronic bronchial catarrh and chronic pleurisy.

**Symptoms.** The respiratory surface of the lungs becomes diminished and the expansion on inspiration is impeded in consequence of the new formation and subsequent contraction of the connective tissue. Hence, difficult respiration is noted in cases of a somewhat more extensive affection; it is particularly noticeable during work and leads to rapid tiring out of the animal. The signs of chronic bronchial catarrh are also usually present, such as a dull, weak cough, scanty, usually sharp (dry) râles, or purring or whistling sounds, etc. They are often noticeable only after deep inspiration, after exercise, after coughing, or after one has previously closed the nostrils of the animal. Disturbances of nutrition become marked later on.

On careful percussion, particularly on corresponding places of the two sides of the thorax, one can usually make out a more or less distinct dullness, occasionally tympanitic sounds over the anterior and inferior portion, as a rule behind or below the scapula. The vesicular breathing is weakened in the area of dullness or is entirely absent; in other cases one may have, on the contrary, bronchial breathing, or intensified, drawn respiratory sounds. In extensive shrinking of the pulmonary tissue many pulmonary capillaries become obliterated; consequently the second pulmonary sound is intensified.

The disease runs its course without fever, and this is important as a point of differential diagnosis with reference to acute pulmonary consolidation. However, bronchial catarrh,



which is scarcely ever absent, occasionally leads to a transitory elevation of temperature.

Lardaceous pneumonia of horses at first leads to diminished appetite, listlessness, cough, emaciation, and to gradually increasing acceleration and difficulty of respiration, with the characteristics of broken-winded trouble. Only after several weeks does percussion reveal, particularly in the upper portions of the thorax, more or less distinct dullness and diminution or absence of the vesicular breathing sounds, and the presence of râles. Bang believes that a chronic alveolar pulmonary emphysema is probably always formed. Fever and acceleration of the pulse come on only after the disease has lasted a long time or after very severe service. The course of the affection is insidious and slow and difficulties in respiration only after one-half to one year, reach such a high degree that the animals can not be used for work.

In bronchopneumonia fibroplastica of horses Grüter observed frequent, strong cough, scanty nasal discharge, accelerated respiration without special use of the muscles of the flanks, unchanged percussion sounds, normal boundaries of the heart and of the lungs.

**Diagnosis.** When dealing with a very slowly progressing pulmonary affection, one must always think of chronic interstitial pneumonia; however, it can only comparatively rarely be diagnosticated definitely. The greatest difficulties in differential diagnosis are offered by echinococcus diseases of the lungs and by pulmonary tumors. The diagnosis is comparatively easiest if the chronic pulmonary disease comes on after the symptoms of acute croupous or catarrhal pneumonia or acute pleurisy have passed off. In cattle and also in other animals it is important to exclude tuberculosis; this can, of course, usually be done by a tuberculin test.

**Treatment** is absolutely non-promising, hence all efforts must be directed toward sparing the animal and promoting a good nutrition, which may retard the further development of the pathologic process. The improvement of the bronchitis will improve the condition of the patient. One might try fibrolysin (for large animals every second to fourth day, one gm., dogs 0.2 gm.; or 10 cc. and 2 cc. respectively of the prepared solution subcutaneously). Animals whose meat is used for human consumption should be slaughtered as soon as their nutritive condition has improved.

**Literature.** Bang, Maandsskr., 1894, V, 218.—Berger, Z. f. Infkr., 1907, III, 356.—Dieckerhoff, W. f. Tk., 1884, 357. Spez. Pathol., 1904, I, 854.—Grueter, Beitr. z. Kenntn. d. Bronchitis chronica des Pferdes, Diss. Zuerich, 1909 (Lit.).—Lubnan, Arch. f. Hyg., 1907, LXIII, 391.—Lüttswager, D. t. W., 1908, I (Lit. on Pulmonary Anthracosis).—Ölkers, Leipzig. Ber., 1906.—Prösch, Leipzig. Ber., 1906, F.—Vansteenberghe & Grysez, A. P., 1905, 787.—Bull., 1906, 695.

## 12. Echinococcus in the Lungs. Echinococcosis pulmonum.

**Occurrence.** Echinococcosis in general, and pulmonary echinococcosis in particular, occurs everywhere. Its frequency, however, varies within wide limits in different parts.



**Most commonly** affected are ruminants and hogs; a few cysts are sometimes found in the lungs of horses. (For more details on the frequency of echinococcosis, see the chapter on echinococcosis of the liver.)

**Etiology.** Echinococci are the cysticerci of the dog parasite *Tænia echinococcus*, the oncospheres of which are probably carried by the blood current from the intestines into the lungs; they change into cysts in the pulmonary tissues; these cysts grow very slowly in size. Infection occurs by swallowing the ova of *Tænia echinococcus* with food or water (on the developmental history and the various forms of echinococcus, see echinococcosis of the liver).

**Anatomical Changes.** In some cases, the lungs contain only a few echinococcus cysts, in others these occur in so great numbers that they cannot be counted, so that the weight of the organ may have become increased considerably (in a case reported by Findeisen, the lungs of a steer weighed seventy-eight pounds). The surface of the lungs then becomes uneven, nodular, and the palpating hand feels the cysticerci in their interior, surrounded by a connective tissue capsule and containing a clear serous fluid. These cysts may be felt as firm, tense, globular nodules, up to the size of a fist. If the cysts are cut into they discharge a serous fluid, and the elastic membrane forming the interior surface can then be peeled off easily from the external capsule. The cysts are sometimes provided with scolices, or they may frequently be sterile, or exceptionally also contain daughter cysts. Small, dry, caseous nodules or mortarlike masses are also often present in foci, which look similar to caseous tubercles, but in which the microscope shows sometimes folded parts of the chitinous membrane, occasionally also hooklets. The pulmonary tissue in the immediate neighborhood of the cysts appears compressed, flaccid, either entirely void of air, or containing very little of it. *Echinococcus multilocularis* has once been found in the lung of a steer. Other cysticerci are usually found in the other organs, particularly in the liver.

**Symptoms.** Only if the invasion is considerable, does the disease manifest itself by difficult respiration, which develops very insidiously, and which, therefore, remains unnoticed for a long time and very rarely takes on a threatening character. With such difficulties of respiration, a cough appears; it is frequently heard after short intervals, usually weak, dull, sometimes even hardly audible. Difficulties of respiration and cough may suddenly increase without any apparent cause (rupture of a cyst into a bronchus or into the pleural cavity). Sometimes exhaustion and overheating bring about an aggravation of the condition. In case of rupture of a cyst, a watery fluid is sometimes discharged through the nose, in which shreds

of the chitinous membrane of the ruptured echinococcus can be seen (Bolz).

The percussion sound remains normal if only small cysts are present or if the cysts are limited to the more central pulmonary portion. If they are more numerous and larger, circumscribed areas of dullness are found in various portions of the thorax. The percussion sound is more rarely tympanitic and the cracked-pot sound may be heard in animals with a more yielding thorax, because cysts may be crowding each other or may be situated near the pulmonary surface. Metallic sounds may possibly be heard over cysts which have become empty. The areas of dullness exhibit diminution or absence of vesicular breathing sounds. Also whistling, purring and other noises. A peculiar noise called "quurksen" by Harms is characteristic for the disease, which noise is claimed to be identical with the sound elicited on rolling and pressing a lung containing echinococcus cysts. Since it is impossible that a noise arises in the cyst synchronously with respiration, such noises must arise in the bronchi. They are simply catarrhal noises and cannot be characteristic of echinococcus of the lung.

The general condition of nutrition of the animals remains satisfactory for a long time. However, when the disease of the lungs reaches a high degree, and when it is associated with echinococcus of the liver, the animals suffer later on in their nutrition. The affection takes an afebrile course.

**Diagnosis.** Only severe cases can be diagnosticated with more or less probability, especially in cattle. One of the most important points to be observed is the presence of severe respiratory difficulties, while the general nutritive condition of the animals shows only very insignificant disturbances. A concomitant increase in the size of the liver must direct suspicion toward echinococcus infection. In special cases one might make use of the complement-fixation test which has been found reliable in animals by Weinberg and Vieillard; in man by Putzu. Cough is more powerful, frequent and convulsive—at least in the beginning of pulmonary tuberculosis; the temperature is elevated. There are also symptoms of a more or less extensive bronchial catarrh and the nutrition of the animals suffers at a comparatively early stage. Pulmonary tuberculosis is distinguished by a more extensive dullness, by bronchial breathing and by a more rapid development which is accompanied by fever.

**Treatment.** There is no successful treatment of the disease; hence early slaughter of the animals is advisable, because otherwise their value is decreased in consequence of emaciation which will come on.

**Literature.** Bolz, W. f. Tk., 1907, 28.—Martin, *Rev. vét.*, 1907, 734 (Lit.). (Also see literature on echinococcus of the liver.)







### Other Parasites in the Lungs.

**Distomatosis of the lungs** is frequently seen in cattle, rarely in sheep, exceptionally only in hogs, buffaloes, horses, asses and dromedaries. The invasion of the lungs is brought about in such a manner that hepatic distomas get into the hepatic and from there into the pulmonary vessels. Globular cysts from walnut to apple size are then formed in the pulmonary tissue, preferably in the posterior cutaneous lobe, which have a fibrous, sometimes calcified capsule, and may, in sheep or young cattle, be surrounded by hemorrhagically infiltrated tissue. In the yellowish or olive green contents of the cysts the ova of distoma are found and occasionally one, exceptionally two of the liver flukes.

Lung distomatosis usually does not lead to any clinical symptoms; occasionally there may be cough, possibly also a circumscribed dullness on percussion, also râles. Emaciation occasionally occurs, but it is then probably due to a simultaneous distomatosis of the liver.

**Lung pentastomosis** caused by *Pentastomum denticulatum* or *Linguatula denticulata* has been observed in a few cases, viz., in the goat (Gerlach, v. Ratz); in the roe (v. Ratz), and in cattle (Hermann, Lungwitz). The larvæ of pentastomum burrow canals into the pulmonary tissue and frequently also into the pleuræ; numerous hemorrhagic foci are formed thereby and the animals may die from cachexia (v. Ratz).

**Worm-nodules and cysticerci**, sometimes seen in the lungs of horses and cattle, are without clinical significance (see liver cysticercosis concerning the occurrence of *Cysticercus tenuicollis*).

**Literature.** Lungwitz, Z. f. Flhyg., 1893, III, 218.—Neumann, Mal. paras., 1892, 560.—v. Ratz, Chl. f. Bakt., 1893, XII, 329.—Régnier, Rev. gén., 1908, XI, 131.

### 13. Neoplasms of the Lungs. Tumores pulmonum.

Tumors of the lungs are not very rare; they are, however, less important from a clinical standpoint, because it is very difficult to diagnose them. Most frequent are carcinomata and adenocarcinomata (especially in dogs), then sarcomata and melanosarcomata (in horses) as a rule as metastatic neoplasms. Fibromata, chondromata, lipomata, osteomata, dermoid cysts are occasionally encountered. Botryomycomata are sometimes found in horses.

The symptoms which might be detected in the living animals are on the whole very indefinite in nature, because usually they refer only to a diminution of the respiratory surface; possibly also to an infiltration of parts of the pulmonary parenchyma with bronchial catarrh; the clinical picture then may be similar to chronic interstitial pneumonia. Sometimes tumors may press upon the heart or upon the larger blood-vessels and may thus bring about congestion in the territory of the jugular vein with hydropic symptoms (see compression of the heart, Vol. I), or there may be compression of a larger bronchus. Very gradually increasing dullness, eventually in combination with bronchial breathing sounds, confined to a certain portion of the thorax, may be observed and may give some information concerning an existing dyspnea. Intrathoracic tumors in smaller animals may be recognized easily in the

Roentgenogram and even in larger animals if they are tolerably large and situated back of the shoulder.

However, a diagnosis of tumor can be made with a good deal of probability only if a primary neoplasm can be detected in the body, if the pulmonary affection can be referred to it and especially if a gradual enlargement of the axillary glands and those at the aperture of the thorax can be observed. Metastases in the lungs are particularly frequent in carcinoma of the thyroid in dogs.

Solitary pulmonary tumors may possibly be removed by operative procedure (Parascandolo, A. f. Tk., 1902, XXXIII, 138; Pansini, Oe. M., 1907, 289).

Proliferation of the alveolar epithelia in the smallest bronchi leads, frequently in sheep, not uncommonly in adult cats, to the formation of nodules of the size of a hazelnut to an apple. They are grayish-white to reddish-gray in color, of homogeneous lardaceous consistency and occasionally become confluent to form larger tumors. These formations have been called "multiple adenomata" by Eber and Prösch, and "polyadenomata bronchiale" by Ball. They appear to arise in connection with a bronchopneumonia in sheep (Prösch) and they do not seem to lead to any disturbance of health. Ball, however, saw in cats accelerated and difficult respiration, cough, a gradual increase of the size of the chest (emphysema of the lungs) without any disturbance of the general condition of nutrition (on bronchopneumonia fibroplastica, see page 156), (Ball, J. vét., 1907, 71; Eber, Z. f. Tm., 1899, III, 161; Prösch. Leipz. Ber., 1906, 7).



## SECTION V.

### DISEASES OF THE PLEURA.

#### 1. Inflammation of the Pleura. Pleuritis.

**Occurrence.** Pleuritis is a common disease of domestic animals. It usually occurs as a secondary affection, especially after pneumonia, more rarely as a primary disease. In horses, which are most frequently affected, pleuritis develops preferably after equine influenza; in ruminants as part of the clinical picture of a hemorrhagic septicemia, in cattle also after pneumonia and in its chronic form on the basis of tuberculous infection. According to Cadéac, serofibrinous pleuritis in dogs is tuberculous in nature in nine cases out of ten. The cases of pleuritis which the authors have seen in dogs almost always developed in consequence of tuberculosis. In fowls, pleuritis is said to occur frequently in combination with peritonitis (Guittard).

**Etiology.** The true cause of a pleuritis is generally an infection, while external influences only play a rôle as exciting or predisposing causes. The disease is often found in septicemic affections due to the bacillus bipolaris. Aside from this microbe, there are frequently concerned the pyogenic bacteria, the tubercle bacillus, the influenza bacterium, more rarely the bacillus mallei, also, in hogs and cattle, the bacillus pyogenes; Piana saw a case of pleuritis in a dog caused by the leptothrix buccalis; the authors and others saw pleuritis in dogs due to streptothrix (actinomyces) canis. Eisenmann saw cases of pleuritis following chronic hog erysipelas. Pleuritis frequently follows contagious nasal catarrh of rabbits (see page 15). Other bacteria than those mentioned may occasionally be the cause of pleuritis.

The invasion of bacteria which cause inflammation of the pleura occurs in a variety of ways. After perforating wounds of the thorax pyogenic bacteria usually enter the thoracic cavity and produce a purulent pleuritis. Traumatic injuries to the thorax, such as a kick, contusion, a fall upon the chest, subcutaneous fracture of the ribs, which may occur without injury to the skin, give rise to the invasion of bacteria, either directly from the contused spot or through the blood current. As a

rule, however, bacteria do not invade the pleura under these circumstances, but the injured tissues may produce an inflammation by chemotactic action, which is limited to the injured territory. All these traumatic insults may cause injuries to the lungs and may cause the invasion of bacteria from the inhaled air into the pleural cavity. More dangerous are internal injuries produced by fragments of bone or other bodies which penetrate from the esophagus or stomach into the thoracic cavity.

In the great majority of cases pleuritis develops in connection with an inflammation of the immediately adjoining organs, especially the lungs, in such a manner that the bacteria causing pneumonia get to the surface of the pleura with the lymph current or else the inflammation spreads by continuity. Pleuritis very frequently follows upon croupous pneumonia, more rarely catarrhal pneumonia; but in some epizootics almost every case of bronchopneumonia leads to pleuritis. Equine influenza is accompanied in almost every case by an intense pleuritis. In purulent pneumonia and gangrene of the lungs pleuritis may occur even without any breaking through of the pulmonary abscess or dangerous focus. Tuberculosis and pulmonary glanders, also bronchial catarrh, may furnish the starting point for pleuritis.

Diseases of the other neighboring organs more rarely lead to a secondary pleuritis. Among them are to be mentioned purulent mediastinitis in the course of strangles, rupture of an esophageal diverticulum or perforation of the thoracic portion of the esophagus by a foreign body, acute and frequent traumatic pericarditis, which occurs particularly in cattle, caries and necrosis of neighboring bones, especially the ribs and sternum, acute inflammation of the peritoneum and of the abdominal organs.

In the course of acute general infectious diseases pleuritis likewise is not uncommon. In this respect the diseases ought to be first mentioned which are caused by the bipolar bacilli (see Vol. I) which may cause pleuritis without a pneumonia existing simultaneously. In the course of pyemia, in rheumatic affections of the joints and of the other serous membranes, in variola and so forth, pleuritis occasionally makes its appearance.

Pleuritis is caused exceptionally by chemicals (bacterio-toxins, constituents of urine retained in the blood in nephritis); mechanical irritation (tumors of the pleura or neighboring organs) in very rare cases by tapeworm, pleurocercoides Baillet (Lefèvre and Guérin), *Cysticercus tenuicollis*.

Cold frequently plays a certain rôle, aside from the causes mentioned; its influence may be very obvious, so that it is easy to understand why cold was formerly considered the exclusive cause of primary pleuritis. The majority of authors (Friedberger & Fröhner, Kitt) believe even to-day in the occurrence



of pleuritis solely produced by cold. The present authors look upon cold as merely an exciting, predisposing cause which makes possible the invasion of the pleura by bacteria living in the animal body or gaining access accidentally or causing a general infection. It is self-evident that the predisposing influence of a cold may be so important that without this factor bacteria would not be able to manifest their pathogenic effect. Cadéac is of the same opinion, and Dieckerhoff believes that infectious bacteria play a rôle in connection with taking cold.

The fact deserves mention that Laborde and Trasbot succeeded in producing a serous inflammation by the intrapleural injection of very small amounts of the serous exudate from cases of rheumatic pleuritis, while the injection of pure, indifferent fluid had no effect whatever. Duviolsart observed in sheep, shorn during the winter, numerous cases of sickness which began on the second day after shearing; some cases developed even after the animals had been brought to a warm place, proving the infectious nature of the affection. (These were evidently cases of hemorrhagic septicemia.) Immelmann's observations among lambs were probably of a similar kind (see page 139). There are no more recent reports about pleuritis due to cold (compare the veterinary reports of the Prussian army).

Influences which weaken the organism, such as overexertion, long-continued railroad transportation, etc., predispose animals to infections in general and to pleuritis in particular.

If there is no disease of any other organ and no general disease which might be the cause of the pleuritis, the latter is designated as a primary pleuritis in contradistinction to secondary pleuritis, which may be referred to another primary disease.

**Anatomical Changes.** The acute inflammation begins with an arterial hyperemia and the surface of the pleura simultaneously becomes rough, lusterless and dry. Blood-plasma is extravasated and also a larger or lesser number of white blood corpuscles; fibrin is deposited in the exudate which covers the surface like a spiderweb or veil, sometimes in somewhat thicker deposits; it is very friable and loosely adherent (pleuritis fibrinosa s. sicca). In other cases, the inflammatory process leads to the formation of an abundant serous and fibrinous exudate (pleuritis seroso-fibrinosa). The serous exudate is sometimes rather clear, yellowish or reddish, possibly greenish (pl. serosa); at other times more or less cloudy, in consequence of suspended flocculi of fibrin and cells; then the pleura is covered with thicker, friable, moist, loose, yellowish fibrinous membranes.

The amount of the exudate varies very much. In the pleura of horses one usually finds 15 to 20 liters of fluid, sometimes much less, at other times much more. (Gosier found 60 liters in one case, Holmes 155 pounds of fluid); the average amount in dogs is  $\frac{1}{2}$  to 5 liters; in hogs 2 to 10 liters (Friedberger & Fröhner).

The thickness of the fibrinous pseudomembrane varies between some millimeters to several centimeters. Its surface is generally uneven and villous. In cases with a very rapid course the amount of fluid exudate usually predominates, in protracted cases the amount of fibrin.

In some cases, the exudate becomes purulent (pl. purulenta s. pyothorax, s. empyema), but it may then also contain



fibrin membranes which cover the affected portion of lungs with fibrino-purulent deposits. Occasionally there may be an extravasation of numerous red blood corpuscles, or the exudate assumes a more or less hemorrhagic tinge (pl. hemorrhagica), while the entrance of saprophytic bacteria causes putrefaction and an ichorous condition of the exudate (pl. ichorosa).

Under the microscope the serous exudate shows few endothelial cells, generally in a condition of fatty degeneration, and numerous leucocytes; the fibrinous coagula contain leucocytes in a reticular matrix. The purulent exudate consists preferably of intact or fatty degenerated leucocytes; while the ichorous exudate contains much granular detritus, fat droplets and shreds of tissue. The chemical analysis of the exudate shows albuminoid substances in larger amounts; 3 to 6% in the sero-fibrinous exudate, and according to Boiteux even 6 to 8.5%.

When there is a considerable mass of fluid in the pleuritic cavity, the lungs, which are already collapsed, are further compressed in proportion to the amount of fluid present; they therefore become gradually smaller and poorer in air. Those portions of lung which are in the neighborhood of the exudate become completely void of air, flaccid and of fleshy consistency; the cut surface appears uniformly dark red (compression atelectasis, splenization). When the exudate is abundant, the mediastinal lobe in horses may undergo a twist outward and backward, which displacement, according to Dieckerhoff, always leads to death, because it prevents the absorption of the exudate. (Mathis found in a dog a complete torsion of the right posterior lobe.)

**Chronic pleuritis** begins with the formation of vascular granulation tissue (pleuritis granulosa), which gradually becomes fibrous and cicatricial (pl. fibrosa s. productiva) or adhesions form between the pleura costalis and pulmonalis (pl. adhaesiva).

Papillomatous granulations occasionally grow out of the tissue of the pleura; Hutyna reported a case where they were very high, branching, and so numerous that they formed a mass the size of a child's heart. Cadot and others have seen the formation of similar granulations on a tuberculous base. Kowalewsky described a case of pleuritis ossificans in a cow.

A fluid exudate is likewise not rare and is either purulent, and in this case surrounded by a connective tissue capsule (preferably in the presence of bacillus pyogenes or of foreign bodies), or serous or hemorrhagic. In horses, peculiar cases of serous, chronic pleuritis are observed where the serous membranes contain a large amount of serous or slightly purulent fluid and where the pleura is studded with thick, fibrinous deposits containing numerous small abscesses. (Streptococci pleuritis, Bang.)

**Symptoms.** The initial stage of pleuritis leads to variable symptoms, according to whether the disease is primary or secondary.

Primary pleuritis usually begins with the non-characteristic symptoms of a general disease. The sick animals gradually lose their appetite, they become dull, their fur looks bristly, they have attacks of chills, an anxious facial expression and avoid every motion. The temperature is elevated from the start. The chilliness ceases after a few hours, but with the hand placed on the thorax one feels occasionally fibrillary trembling of the intercostal muscles (Trasbot). The number of pulse beats is, as a rule, increased 50% and the pulse is weak. Some horses show at this stage also slight symptoms of colic and they give evidences of pain (cutting) in the chest wall. In secondary pleuritis these symptoms are preceded by those of the primary disease and they may be clouded, partially or completely, by the symptoms of the primary affection.

The respiration is markedly accelerated and superficial in every case of pleuritis; it is also usually of the abdominal type, the patients hold their ribs as quiet as possible and use the diaphragm in preference. Exceptionally, however, especially in inflammation of that portion of the pleura which lies near the upper surface of the diaphragm, breathing is of the costal type.

Palpation often shows an increased tenderness of the wall of the thorax, the animals try to withdraw when pressure is made upon the intercostal places, and they betray pain by crying out or groaning. The tender territory is usually behind the elbow and extends a certain distance backward and upward. On palpation, and better still, with the flat hand, one sometimes feels a trembling of the thoracic wall, which is synchronous with respiration and indicates a roughness of the involved portions of the pleura.

Cough is not observed in all cases, and some, even severe ones, may run their entire course without any cough. When it is present, it is always weak and the animal tries to suppress it. But it may easily be brought on artificially by percussion of the thorax.

The percussion sound is normal in the beginning if the superficial portions of the lungs are not affected; in large animals a certain amount of fluid exudate may collect in the lower portion of the thorax without changing the normal percussion sound. Fibrinous membranes are almost never formed to such an extent that they will materially dull it.

The vesicular breathing is diminished owing to the slighter lung movements; occasionally also in consequence of the presence of thicker fibrinous membranes. Friction sounds are frequently heard; these are sometimes soft, sometimes grating or crackling. They are rarely strong enough to be heard at a distance, and are usually heard by the ear placed over the affected area; in smaller animals it is often necessary to press the stethoscope down firmly. The sounds occur synchronously with the respiratory movement, and they are sometimes



heard only during inspiration, eventually only toward its end; sometimes both during inspiration and expiration. Exclusive expiratory sounds are rare. The sounds are frequently interrupted for short periods.

A deviation from the behavior of the friction sounds occurs when they are caused by rough portions of the pleura which are in immediate contact with the parietal layer of the pericardium (pericarditis externa); then the possibility exists that the friction sounds are synchronous with the contractions of the heart muscle. Under these conditions the friction sounds depend more upon the apex-beat (pleuro-pericardial sounds), but they are also influenced by the respiration and either accompany the inspiratory or the expiratory movements synchronously with the heart sounds, while they may also be observable during the pause of the heart sounds, but with the respiratory movements. Such sounds are, of course, only heard in the cardiac region and more frequently on the left than on the right side.



Fig. 25. Horizontal line of dullness in pleurisy with fluid exudate (Tuberculosis).

In pleurisy with a fluid exudate the picture so far described is altered very considerably. The respiratory movements are now usually less accelerated, but are deepened and forced in proportion to the amount of fluid present (flank breathing). During inspiration the ribs are elevated and there is a distinct trembling of the vertebral column and of the whole rump at the beginning of expiration. Expiration takes place in two stages and produces a groove parallel to the costal arch at the beginning of expiration; the flank depressions become prominent simultaneously, and the lumbar portion of the vertebral column occasionally curves upward. In very severe cases, a pumping respiration is observed. The patients also manifest other symptoms of dyspnea.



When the amount of exudate is very great, changes in the shape of the thorax may appear. In smaller animals an enlargement of the affected half of the chest is occasionally seen distinctly, while the intercostal spaces of larger animals may be flattened or even protruding. In some cases, especially in larger animals, non-painful edemata are seen on the anterior thorax, the lower thorax or even on the abdomen.

The tenderness on pressure of the intercostal spaces decreases or disappears entirely in the region of the fluid exudate. It persists, however, in the higher portions of the thorax if these are the seats of dry pleuritis. The painful character of the cough persists also in the further course.

Percussion shows a horizontal dullness either on one or both sides of the chest (Fig. 25), with a considerable increase



Fig. 26. Horizontal line of dullness in pleurisy in sitting posture of the animal. (The same as in Fig. 25.)

in resistance. Parallel with the boundary of complete dullness and extending some centimeters upward there is some dullness, followed by a normal sound. If, however, the dullness extends over the lower half of the thorax, the percussion sound of the other half is diminished, because the high column of the fluid also interferes with the vibrations of the free portion of the wall of the thorax. When larger animals lie down, the area of dullness becomes diminished or it may occasionally disappear entirely and it changes also in smaller animals with changes of position of the body (Fig. 26). In case of an en-

capsulated exudate the horizontal boundary of the area of dullness is absent and it does not change with the changing position of the body. Tympanitic sounds are observed only in those rare cases in which, with a decrease of the fluid, the superficial layer of the erstwhile collapsed lung portions again contain air. In smaller animals tympanitic sounds are heard also over those portions of the thorax which are not occupied by fluid; the same condition is also observed in a lateral position of such animals. The cause of the tympanitic sound in smaller animals is furnished in a comparatively considerable diminution of the sounding column of air enclosed in the thorax.

Auscultation shows absence of friction sounds in the area of dullness; further upward they are, however, commonly heard later on. The other respiratory sounds are likewise much diminished or absent in the area of dullness, and along the upper boundary indefinite or bronchial breathing is heard in many cases. If, however, the lung has become adherent at the onset of the disease or previously, bronchial breathing is heard also in the area of dullness. The vesicular breathing is intensified above the area of dullness and on the healthy side.

The apex beat is weakened on the diseased side and the heart sounds are duller and weaker. Toward the termination of severe cases the symptoms of cardiac weakness appear.

The temperature (Figs. 27 and 28) does not give a typical curve. Great variations of temperature are usually seen in the later course of the disease. Remittent or continuous fever in less acute cases is occasionally interrupted by fever-free intervals. Very high fever is usually seen in purulent or ichorous pleuritis.

Continuous standing during the course of the disease is frequently observed in horses. If the animals lie down exceptionally they rest on the sternum or on the healthy side, as do other animals in the beginning stage of pleurisy, in order to protect the tender portions of the thorax against pressure; after an exudate has been formed they lie on the diseased side, so that the healthy portions of lung have a chance to expand. If animals in this stage are, during examination, placed upon the healthy side, the dyspnea is at once markedly increased and the animals may even suffocate, because the healthy parts of the lungs are compressed by the body weight, and the affected portions by the exudate.

The appetite is capricious and only becomes normal after recovery has set in. If the disease lasts for a long time the animals become markedly emaciated.

The amount of urine is diminished in the beginning; the amount of chloride of sodium diminishes markedly as the exudate forms, while its absorption leads to an increase of urine and of its chloride of sodium contents (urinary crisis). Al-



buminuria is not uncommonly present during the course of the disease.

**Complications** are frequent, even if the primary diseases followed by a secondary pleurisy are left out of consideration. Very important, as a complication, is pericarditis, because it may lead to complete cardiac exhaustion. Delacroix found embolism in the pulmonary arteries in a horse which died suddenly during recovery from pleuritis; the origin of the embolism was found in a thrombotic, bent posterior vena cava. An abscess is sometimes found in the wall of the thorax in purulent and ichorous pleuritis; the abscess breaks outside, or it opens into one of the thoracic organs, into the lungs, or through a cavity of the latter into a larger bronchus, into the trachea, pharynx, mediastinum, etc.

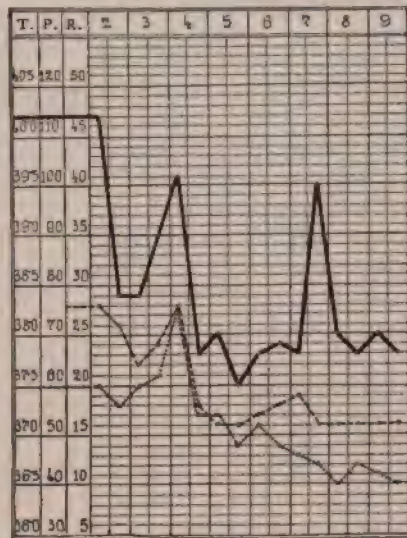


Fig. 27. Fever curve in primary pleurisy in a horse. Recovery.

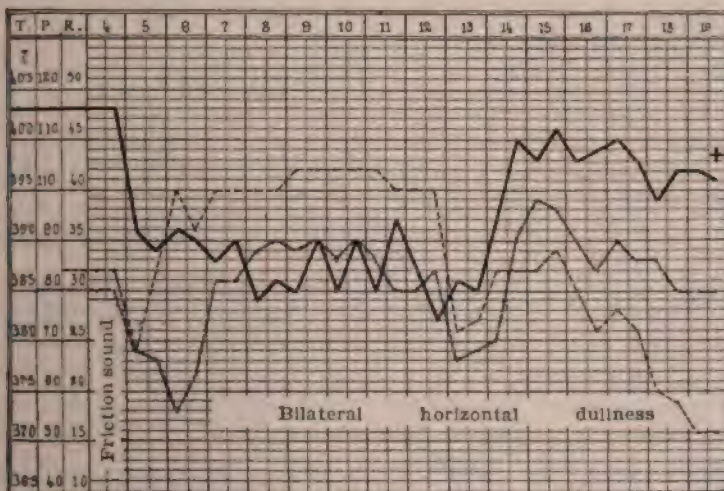


Fig. 28. Fever curve in secondary pleurisy in a horse, (following upon croupous pneumonia).

According to Guittard, **pleuropneumonia of fowl** at first leads to listlessness, diminished appetite, retarded digestion, and after several days to great prostration, the comb becomes pale and the respiration



forced. In the further course of the disease the animals die quickly after diarrhea has set in. An exact diagnosis can only be made on post-mortem examination.

In the beginning and often during its whole course, **chronic pleuritis** does not lead to very prominent symptoms, which become marked especially during exercise. If there are extensive adhesions, the vesicular breathing is less marked, but the percussion sounds remain normal. In the presence of serous or more purulent exudates in the thoracic cavity, after certain occurrences, for instance after hard work or after a cold, one observes acute exacerbations which, in addition to already existing areas of dullness and diminution or absence of breathing sounds, lead to tenderness of the thoracic wall, marked dyspnea, dry, rough cough and moderate elevation of temperature. Displacement of the heart by the exudate leads to change of the apex beat and in the intensity of the different heart sounds in various places of the cardiac region; disturbances of circulation may also be present (stronger filling of the veins, presystolic venous pulse, edematous infiltrations). (See compression of the heart, Vol. I.)

Disturbances of nutrition appear sooner or later. The hair becomes lusterless and ruffled, the skin dry and less elastic; emaciation gradually reaches a high degree, the production of milk diminishes, the symptoms of cardiac weakness come on and the animals die exhausted and emaciated.

**Course.** Very mild cases of fibrinous pleurisy sometimes run a very rapid course, occasionally one of a few hours only. The symptoms may be of a very minor character, very insignificant tenderness, soft friction sounds; otherwise nothing abnormal can be detected in the chest. These symptoms may entirely escape notice, as is proved by the well-known fact that thickening of the pleura and adhesions are not infrequently found in animals who have always been under observation and who never showed symptoms of pleurisy.

The formation of an abundant fluid exudate often requires a long period of time; sometimes, however, the pleural cavity may become filled within a few days to over one-half of its space. The increase of the exudate usually occurs periodically and is accompanied by elevation of temperature. Occasionally the exudate only increases to such an extent that life is not endangered for several months.

Pleurisy with exudate may end in complete recovery, especially when the exudate is not very fibrinous or is purely serous. In the majority of cases chronic tissue changes, such as thickening and adhesions, remain as permanent residua. Whenever the quantity of the exudate is considerable, its absorption requires several weeks and is interrupted by temporary relapses. Adhesions which have been formed may sub-



sequently interfere with the proper movements of the lungs. Most dangerous in this respect are adhesions at the lower posterior thirds of the lungs, or the lower posterior margins. Animals with adhesions in these parts usually suffer for the rest of their lives from shortness of breath, even if their general state of nutrition is good. Adhesions of the lungs may also bring it about that the portion which is fixed in an abnormal place is unable to return to its normal place after the exudate is absorbed. The neighboring portions must then expand more extensively, the heart may be displaced toward the affected side and the thoracic wall may eventually be depressed.

Chronic pleurisy does not terminate in complete recovery, particularly since the compressed pulmonary tissue becomes permanently solid, on account of the collapse of the alveolar walls, and on account of the connective tissue proliferation. Such animals are easily tired out when worked, their nutrition is not good, they cough periodically, breathe with difficulty and the presence of an exudate in the lower portion of the thorax can be demonstrated objectively.

Chronic pleurisy sometimes develops as such from the start and only reaches a high degree after months. According to Rigot and Cruzel, this form is particularly common in cattle, and as Lafosse and Trasbot state, particularly after milder, but frequently recurring, colds.

A fatal termination of pleurisy occurs in acute cases usually after two to three weeks, in consequence of suffocation or exhaustion. Before death the sick animals show great dyspnea, the nostrils are convulsively dilated in each inspiratory act, the ribs are extensively elevated with a simultaneous elevation of the spinal column and the mucous membranes are cyanotic. The animals finally fall down, try several times to get up again, kick around the floor and die in convulsions. Death sometimes comes on quite unexpectedly with the symptoms of cardiac weakness during an attack of suffocation. In some chronic cases the animals succumb completely prostrated.

**Diagnosis.** The only pathognomonic sign of pure fibrinous pleuritis is the friction sound heard synchronously with the respiratory movements. If, however, in the course of a disease which is usually followed by pleurisy, tenderness of the intercostal spaces come on, one is justified in thinking of the probability of a beginning pleurisy. Rheumatism of the intercostal spaces (pleurodynia) is very rare and can be distinguished from pleurisy, even in the absence of friction sounds, on account of marked general disturbances which are present in pleurisy, by the accelerated and weak pulse, acceleration of the superficial, so-to-say trembling, respiratory movements. Affections of the ribs (fracture, periostitis, caries) are characterized by swelling and tenderness confined to the ribs, as well as by the absence of general symptoms.



Pleuritis with fluid exudate is characterized by the following clinical picture: marked horizontal dullness, often on both sides, strong increase of resistance and the absence of respiratory sounds in the area of dullness. Exploratory puncture may be necessary to clear up a doubtful case. Croupous pneumonia is differentiated by the presence of an area of dullness which is bounded upward by a curved line or which rises or falls posteriorly, and by bronchial breathing which is audible at least from time to time. The initial symptoms are also very different. The onset of pneumonia is characterized by a reddish yellow discoloration of the mucosæ, eventually by a rust-colored nasal discharge, by deep and difficult respiration and by a strong pulse; in the beginning of pleurisy the respiration is much accelerated, superficial, almost trembling; the pulse is hard and small and the hand placed on the chest wall can detect a muscular tremor. The fever curves of the two diseases differ materially. The course of the fever in pleurisy is similar to that in catarrhal pneumonia; however, a continuous area of dullness is either absent or it is shaped as in croupous pneumonia.

The difficulties increase, however, when it must be decided whether pneumonia and pleurisy exist simultaneously. The shape of the area of dullness is then not decisive, because fluid below and infiltrated lung floating on it above may give a curved boundary line of the area of dullness. One also hears bronchial breathing sounds in the floating lung especially near the upper boundary of the area of dullness, as it is often found in croupous pneumonia. The behavior of the apex beat and of the heart sounds often is decisive; a diminution in their intensity points to pleurisy. Important also is the presence of edema on the inferior thorax, which does not occur in pneumonia.

The differential diagnosis between pleurisy and pleuropneumonia in cattle may be difficult when pneumonia consolidation exists simultaneously with pleurisy. The concomitant circumstances, such as the possibility of importation of the contagion must then be considered. A suspicion of the existence of pleuropneumonia appears justified where subacute or chronic pleuritis or pleuropneumonia are found and where the possibility of a contagion cannot be excluded.

Pleuritis may be distinguished from pericarditis by the fact that in the former case the apex beat is not as much intensified and as rhythmic as in the latter case; the friction sounds in pleuritis are usually heard on one side only, they are influenced by the respiratory movements, and friction sounds and tenderness of intercostal spaces are usually found in other areas aside from that of the heart. Apex beat and heart sounds are almost absent in pericarditis with effusion; this is contrary to what is found in pleurisy.

Anamnetic data and the eventual simultaneous presence



of other pathologic processes deserve full consideration in establishing the particular nature of existing pleurisy, which may depend upon a purulent inflammation of neighboring or more distant organs; purulent or ichorous pleurisy sometimes occurs after penetrating wounds of the thorax. Pleurisy accompanying chronic affections of the lungs (tuberculosis, glanders, actinomycosis, lungworm disease) is usually fibrinous or serofibrinous. In tuberculosis and in cachectic processes in general it may also be hemorrhagic. Assistance in the diagnosis may be afforded in cattle and also in dogs by a tuberculin test. The character of the exudate is best ascertained by an exploratory puncture, which is perfectly void of danger and which should be performed in all doubtful cases.



Fig 29. Sediment of an exudate in serofibrinous pleurisy of a horse. *a*, polynuclear leucocytes; *b*, red blood-corpuscles; *c*, endothelial cells in a condition of fatty degeneration.

The **exploratory puncture** is best made with a hypodermic syringe; its needle is pushed into the area of dullness, into the pleural cavity, best in the sixth or seventh intercostal spaces. In withdrawing the piston of the syringe, fluid often enters even if the needle has been pushed into masses of fibrin. The end of the needle may touch the lung and in this manner we may eventually gain some information about the consistency of the lungs; if the needle has been pushed into the lung parenchyma, we get pure blood or, in the presence of pneumonia, a bloody stained fluid. The fluid obtained by puncture may be used for bacterioscopic examination and for animal inoculation.

The **prognosis** depends upon the etiologic factors, the character of the exudate, the species and the age of the animals affected. Pure fibrinous pleurisy following croupous pneumonia, is not of very grave significance, but the accumulation of a fluid exudate under these and other circumstances is usually an un-

favorable symptom. Cases with high fever, chills and great prostration, with rapid increase of the exudate, usually take an unfavorable course. A guarded prognosis is, however, indicated even in less tempestuous cases, because relapses are common and may lead to a fatal issue. Speedy death must be expected in purulent and ichorous pleurisy. The formation of a hemorrhagic exudate into the pleura under marked acute symptoms also has an unfavorable significance.

In considering the prognosis one must also think of the sequelæ which may materially decrease the value of the animal. Adhesions usually remain after a pleurisy which has lasted three to four weeks; relapses and the formation of an area of permanent dullness exclude the possibility of complete recovery.

**Treatment.** The most important measures are the establishment of favorable hygienic conditions, regulation of diet and nutrition of the animal following the same principles as laid down for the treatment of croupous pneumonia (see page 120). Energetic antiphlogistic treatment is indicated in the beginning of pleurisy when friction sounds are still present. To this end the thoracic wall should be irrigated with cold water, or snow or ice should be applied to the tender region. This must be continued until the sensitiveness, the friction sound, and the high fever has subsided. Good results may be obtained in the beginning with these applications. Inunctions with irritating or acrid substances, based upon the principle of counter-irritation, appear less indicated, although they were formerly much practiced.

French veterinarians even today extensively employ the so-called revulsive treatment by irritating inunctions applied to the thorax from the start and alleged to stop the inflammation. Venesection is practiced for the same reason. Trashot recommends the following treatment in acute cases; in the very beginning venesection, with the removal of an amount of blood in proportion to the existing plethora of the animal, then energetic treatment with preparations of mercury, salicylates and digitalis.

In the further course, beginning on the second or third day, moist, warm applications may be made. Energetic inunctions may be serviceable in delayed absorption, viz., spirits of mustard, 6-10%, chloroform liniment (chloroform 10.0, liniment volatile 40.0). These are to be applied twice or thrice daily over the area of dullness, while more intensely acting drugs, such as cantharidin, croton oil, euphorbium ointment, etc., should rather be left alone. Priessnitz' applications may be employed two to three hours after the inunctions.

NOTE.—In human medicine applications of green soap have proved very efficient means of stimulating delayed absorption of pleuritic exudates (see Report from the Winyah Sanatorium, Drs. K. and S. von Ruck, Asheville, N. C., 1911, page 4). This should prove of value in animals also. The green soap is applied by friction over the affected side after the skin has been washed with ordinary soap and hot water. As a rule the application is made at bed time, is allowed to remain over night and repeated daily, or less frequently, depending upon the tolerance of



the skin. When hyperemia occurs as an effect of irritation, this is allowed to subside before the application is repeated. In many cases the skin is tolerant enough to permit daily applications; in others marked hyperemia follows after its first use in which event the soap must be diluted (Transl.).

Narcotics are to be employed in severe tenderness of the thoracic wall and against tormenting cough. Fever requires interference only when it is very high and when the affection has set in in a very tempestuous manner; then the drugs recommended elsewhere (see page 122) are to be considered.

Diuretics are indicated in the presence of a diminished excretion of urine; powerfully acting drugs should however be avoided. Aside from those medicines which were recommended for croupous pneumonia (see page 122) the following are to be recommended: digitalis (daily 3-7 gm. or to dogs as an infusion 1 : 100 in tablespoon doses, not to be continued longer than one week; liq. potassii acetici (100-180.0 or 5-10.0 per diem, also in combination with digitalis); oil of turpentine (10-15.0 or 1-2.0); calomel (2-4.0 or 0.03-0.05) must be discontinued at once after diarrhea has set in; common salt. To increase absorption the following drugs are serviceable: pilocarpine (0.1-0.2 and for small animals 0.005-0.01 gm. subcutaneously); arecoline (0.08 gm. for horses subcutaneously) and the laxatives, especially the salts. Animals with cardiac weakness should not receive pilocarpine or arecoline.

The most reliable procedure to diminish the amount of the exudate is puncture of the thorax (thoracocentesis), an interference absolutely void of danger if performed with aseptic and general precautions. Thoracocentesis is indicated especially when there is immediate danger of suffocation and in purulent and ichorous pleurisy. There exist, on the other hand, numerous observations (Almy, Liénaux, Fröhner, Marek), which show that puncture made repeatedly and immediately after the exudate has formed may bring about a speedy amelioration even if only a small amount of fluid has been removed. The often observed favorable influence, even after the removal of a small amount of exudate, may be explained by a diminution of the pressure exerted upon the blood and lymph vessels of the pleura. (There is hope for the absorption of the exudate then only if the molecular concentration of the latter is not greater than that of the blood plasma. This has been demonstrated in the case of man by Kétli and Torday.) The primary affections, of course, influence materially the possibility of successful treatment in secondary pleurisy. Fröhner is inclined to believe that only those cases offer hope in which the exudate is free from bacteria. A favorable result does not occur after puncture in cases where the lungs have lost the power to expand.

The puncture is best made in the seventh intercostal space but on the right side it may be made in the sixth space; in larger animals 3 to 5 cm. above the costal cartilages. The instrument may be simply a hollow needle provided with a long, slender rubber tube, the free-end of which should dip into sterile water. A trochar (Billroth's trochar is particularly serviceable) is still better adapted which has the canula provided with a stop-cock which should be closed after the withdrawal



of the stiletto; then a rubber tube can be fastened to the trochar. This simple procedure does, however, not always lead to success, because the exudate rarely stands under sufficient pressure to force the fluid out of the rubber tube. It is therefore frequently necessary to employ the aspirators of Dieulafoy or Potain, which will permit the aspiration of fluid out of the thorax and offer the additional advantage of preventing the entrance of air into the thoracic cavity. The entrance of bacteria into the thorax during puncture must by all means be prevented because this would lead to a more serious and more malignant inflammation.

The evacuation of the exudate must always be brought about gradually and slowly in order to avoid sudden changes of the intrathoracic pressure, as these might lead to extensive capillary hemorrhages and paralysis of the heart. The puncture must be interrupted at once if the yellowish exudate becomes hemorrhagic, if the patient is attacked by cough or if the needle touches the lung. If the pulse becomes weak the patient should receive a subcutaneous injection of caffeine or oil of camphor. Occasionally one might try intravenous injections of adrenalin (Barr). After the termination of the puncture horses may receive a subcutaneous (Boucherion) or an intravenous (Almy) injection of 2 or 3 liters of physiologic salt solution. Fairise recommends a subsequent intrapleural injection of  $1\frac{1}{2}$  to 4 liters of a 1 to 1.5% solution of gelatin in order to prevent hemorrhage or a rapid renewal of the exudate. Subsequent dribbling of the exudate into the intramuscular tissue through the puncture in the thoracic wall may be prevented by the use of a finer trochar.

The amount of exudate which may be removed by one puncture depends upon circumstances; if there is intense dyspnea, enough should be withdrawn to remove the latter (in horses 20 to 40 quarts). In cases where the amount of the exudate is moderate and where there is no immediate danger of suffocation, the removal of 4 to 5 quarts in large, and of 250 to 500 cc. in small animals, is sufficient; larger amounts should be removed at one time only if repeated punctures have shown that additional amounts of fluid have not been formed. It is well known that horses can stand, without damage, the removal, at one time, of 30 to 40 or 50 quarts of fluid; however, this may exceptionally lead to the death of the animal, either in consequence of paralysis of the heart (Trasbot, authors' observation) or on account of extensive capillary hemorrhage (authors' observation). These circumstances, as well as the fact that withdrawn fluid may reaccumulate in a short time and so weaken the patient considerably, let it appear advisable not to withdraw very large amounts of fluid at one time.

Simple puncture is not sufficient in purulent or ichorous pleurisy and it is usually not even successful if the thoracic cavity subsequently is irrigated repeatedly with a disinfecting solution (boracic acid 2-4%; thymol, 0.1%; acetate of lead, 1-2%). Bergeon succeeded in a cow by repeated irrigations with the following solution: tincture of iodine, 60.0, iodide of potash, 20.0, boiled water, 10,000 cc. Ordinarily the only procedure that holds out any promise of success is to open the thoracic cavity, and the resection of ribs, followed by cleansing of the chest cavity (Cadéac, Pointot and others).

**Literature.** Almy, Bull., 1901, 128.—Aubry, Rev. vét., 1905, 629.—Barr, M. m. W., 1904, 982.—Bergeon, Rev. vét., 1907, 505.—Duvieuxart, Rec., 1845, 721.—Eisenmann, Monh., 1906, XVII, 97.—Fröhner, *ibid.*, 1905, XVI, 44.—Guittard, Pr. vét., 1908, 1.—Immelmann, Pr. Mt., 1883, 46.—Kowalewsky, Vet. Jhb., 1906, 158.—Lefèvre and Guérin, Bull., 1903, 145.—Liénaux, Ann., 1903, 65.—Mathis, J. vét., 1895, 39.—Moussu, Rec., 1906, 153.—Pointot, Rec., 1901, 28.—Pr. Mil. Vh., 1899-1908.—Trasbot, Dict., 1889, XVII, 291 (Lit.).—Rec. 1892, 425. (See also literature on the primary diseases.)

## 2. Dropsy of the Thoracic Cavity. Hydrothorax.

Hydrothorax occurs as a part of the clinical picture of certain organic and general diseases and consists in the collection of a serous fluid in the thorax without inflammatory changes in the pleura.

**Etiology.** Hydrothorax is usually caused by a congestion in the vena cava and its branches. It is therefore observed in chronic diseases of the lungs; still more frequently in chronic cardiac diseases and is usually associated with ascites, hydro-pericardium and anasarca. The affection is similarly developed in compression of the venous trunks (compare compression of the heart in Vol. I).

A small amount of clear fluid due to cardiac weakness is usually found in the pleural cavity after a prolonged agonal state.

Hydrothorax in other cases depends upon general anemia, hydremia, or indirectly upon a long-standing exhausting disease (glanders, renal disease, echinococcus, carcinomatosis, etc.). The effect of substances which are irritating to the capillary walls may play a rôle in some cases.

No observations have been made in veterinary medicine to decide whether congestion in the lymphatics, particularly in the thoracic duct, may be the cause of hydrothorax.

**Anatomical Changes.** The transudate in the thoracic cavity appears light, or reddish yellow, perfectly clear or slightly turbid; it may contain very fine flocculi of fibrin, occasionally also numerous red blood corpuscles. It contains a considerable amount of albumin; however, the amount of the latter and the specific gravity are less than that of the blood serum. One usually sees few formed elements under the microscope (desquamated, swollen epithelia, a few lymph corpuscles and erythrocytes and some cells in a condition of fatty degeneration). The lungs are compressed in proportion to the amount of the transudate; the pleuræ either show no changes at all, or are somewhat thickened or cloudy. Congestion is usually present.

**Symptoms.** Hydrothorax usually develops uniformly on both sides and the signs pointing to a collection of fluid in the thoracic cavity (see page 166) are found bilaterally. The other signs, especially those of compression of the lungs, are the same as in serous pleuritis; they develop, however, somewhat more slowly, without fever and without tenderness of the thoracic wall. The serous fluid easily changes its place and the area of dullness changes with changes in position of the patient; the upper boundary always remains horizontal. The lower portions of the thorax appear wider in the presence of an abundant transudate and the movement of the ribs also requires the action of the accessory respiratory muscles.

The **diagnosis** depends upon the detection of the primary disease, the presence of other hydropic conditions, the pro-



tracted and variable course of the affection. Exploratory puncture furnishes further proof; the fluid is usually only slightly turbid, the specific gravity below 1016, the sediment contains few white blood corpuscles. Chaleur and Labasque removed the transudate of a horse suffering from compression of the vena cava due to a melanotic tumor, and found black, sand-like masses of pigment.

**Treatment.** The amelioration or aggravation of hydrothorax goes hand in hand with the primary disease toward which the treatment has to be directed. If suffocation threatens, puncture may be resorted to. It may afford temporary relief.

### 3. Collection of Air in the Pleural Cavity. Pneumothorax.

By pneumothorax in the limited sense of the term is meant a pathological condition, when atmospheric air gets between the parietal and visceral layers of the pleura and causes collapse or compression of the lungs. Those rare cases must also be included where other gases get into the pleural cavity.

**Etiology.** Air is enabled to enter into the pleural cavity in consequence of penetrating traumatic injury to the chest wall, on account of the negative pressure in the chest cavity. The lungs then collapse in consequence of their elasticity. Pneumothorax is similarly brought about when a foreign body penetrates into the diaphragm from the omasum; so that the gases in the latter can get into the pleural cavity. Pneumothorax is also frequently observed in consequence of disease of the lungs. Injury to the lungs may occur in penetrating trauma to the thoracic wall, in subcutaneous fracture of the ribs, or through a foreign body aspirated into the air passages. The possibility of rupture of the superficial strata of the lungs also exists when the air in the lungs is suddenly placed under high pressure (cough, violent action of the abdominal walls, drawing of heavy loads, vomiting, great efforts, continuous bellowing, falling, sudden pressure upon the thorax, etc.). Purulent or ichorous pulmonary foci communicating with bronchi, occasionally echinococcus cysts (Deupser, Sperling) may break into the pleural cavity. Perforation of the pharynx may in very rare cases lead to pneumothorax.

**Pathogenesis.** The amount of air or gas which enters depends upon the character of the opening. If the latter is not closed (open pneumothorax) enough air enters to bring about a degree of collapse of the lungs which is seen in cadavers after opening of the thorax. Later on the air left in the lungs is also absorbed and the latter may become entirely void of air. If, however, the opening is of such type that it will open during



inspiration and close during expiration (valvular pneumothorax), then the air pumped in on inspiration will rapidly compress the lungs and expand the wall of the thorax. If the opening closes sooner or later (closed pneumothorax) the lungs will also collapse proportionately to the amount of air entered. But on account of the closure of the opening they will not become compressed, the air is uniformly distributed around the external and lower portions of the lungs, and can be absorbed later on.

With the air streaming in through the wall of the thorax or from the air passages and always with air coming from neighboring organs, bacteria get into the pleural cavity and may lead to fibrinous, more usually to purulent, pleuritis.

**Anatomical Changes.** Air does not stream outward or inward in open pneumothorax after opening of the thoracic wall; it streams occasionally into the chest cavity in closed pneumothorax and streams out with a hissing sound in valve pneumothorax; it may then move a flame held before the opening. If the trachea had been closed air-tight before an injury to the lung, on opening the chest cavity, one can easily recognize the collapse of the lungs as it occurred during life. We also usually find the signs of serofibrinous (hydropneumothorax) or purulent pleuritis (pyopneumothorax). The changes are usually found in one, rarely in both pleural cavities.

**Symptoms.** The symptoms of pneumothorax either set in suddenly or they develop very gradually, according to whether air got into the pleural cavity suddenly or gradually. More or less intense dyspnea develops in consequence of the displacement and collapse of the lung. It often increases gradually and may lead to attacks of suffocation and to a fatal termination, within one-half to one day. Rapid deterioration is particularly seen after bilateral pneumothorax or after one due to the breaking through of an abscess or of a gangrenous focus. Slow ingress of air in other cases leads to much less violent symptoms; these may, in fact, be so slight that they are overlooked and the pneumothorax is only discovered during a physical examination.

The shape of the thorax in valvular pneumothorax may be so changed that the diseased half is more prominent, while in other forms the healthy side or both halves may become prominent. Tenderness on pressure upon the intercostal spaces points to pleuritis or to fracture of the ribs.

In valvular pneumothorax, the percussion sound is atympanic, loud, and possibly somewhat weakened, in consequence of increased tension of the chest wall. In open pneumothorax, the sound may be tympanitic in all species of domestic animals, in the presence of a sufficiently large opening in the thorax wall; if, however, pneumothorax is open toward the air passages and if the opening can not be large, a tympanitic sound is only heard in small animals, because in them only the small column of air



necessary for the production of tympanitic sound will be present under these conditions. In open pneumothorax one commonly also hears the noise of the cracked pot. The percussion sound of a closed pneumothorax, in total or considerable pulmonary collapse, remains atympanitic in large animals; it becomes, however, distinctly tympanitic in small animals, and also in large animals if conditions have brought about the formation of a small column of air.

One usually hears a metallic sound on ordinary percussion, frequently only on auscultating or on percussion with two hard objects; this is, as a rule, audible over the whole of one-half of the thorax. If adhesions have previously been formed, or if rapid closure of the opening has permitted the entrance of only an inconsiderable amount of air, the metallic sound is heard over a limited area only. When there is an open communication of the pleural cavity with the air passages, one hears the metallic sound, even at a distance, in smaller animals after opening their mouth. The percussion sound of the chest may, posteriorly, on the affected side be followed up to the costal arch; the same occurs, however, on the healthy side in consequence of a compensatory distention of the lung on the healthy side.

After the development of pleurisy with effusion, one can demonstrate dullness to a variable extent with an upward horizontal boundary line; here there is no metallic sound.

Auscultation demonstrates conditions which vary from ease to ease. Sometimes one hears no breathing sounds at all in consequence of intense collapse of the lungs; as a rule, however, the breathing sounds penetrate through a not overly thick column of air. One then hears bronchial, possibly amphoric breathing; usually, however, only a metallic sound. If there is fluid and air between the layers of the pleura, one hears metallic splashing with every respiratory movement, which becomes still more intense in sudden movements or in shaking of the animals. In exceptional cases, one sometimes hears dripping sounds.

The heart beat is not felt at all on the affected side, or only faintly; the heart sounds are occasionally accompanied by a metallic note.

**Course and Prognosis.** The described symptoms usually last only a short time, especially if pneumothorax follows an already existing pneumonia or pleurisy; however, even in animals which were previously well, pleurisy usually develops soon and brings about a fatal issue. Pneumothorax from injury or rupture of a healthy lung ends in recovery somewhat more frequently; one due to penetrating injury of the thoracic wall rarely, i. e., when infection of the pleura has not occurred. The air is absorbed in such cases after closure of the injury, and the lungs again expand.

**Diagnosis.** Characteristic for pneumothorax is the metallic sound present over a large portion of the thorax and brought

out by percussion and auscultation. When lung cavities are present, tympanitic and occasionally also metallic sounds are present only in circumscribed places, and they are usually surrounded by an area of dullness. Diaphragmatic hernia might give rise to errors in diagnosis, if parts of the stomach or loops of intestines have prolapsed into the thorax. But the tympanitic or metallic sound which may be present in the posterior and inferior portions of the thorax reaches a high intensity; its pitch varies with the peristaltic movements and, if these are arrested on account of incarceration, the symptoms of intestinal occlusion are present. The character of the fluid which might be present can only be ascertained by an exploratory puncture.

**Treatment.** In closed pneumothorax, the air ought to be removed. An ordinary trochar is not well adapted for this purpose, because more air might be let in by it. Therefore, one has to use a trochar with a stopcock or, still better, a Dieulafoy aspiration apparatus. In the absence of such devices, a simple trochar or hollow needle may still be serviceable if one attaches to it a rubber tube with a clamp. In pneumothorax which opens outward the opening must first be closed.

The operation recommended for purulent pleurisy might be tried in purulent pneumothorax.

**Literature.** Darrow, *Rec.*, 1902, 482.—Deupser, *B. t. W.*, 1890, 321.—Dorsprung-Zelibo, *Vet. Jhb.*, 1902, 126.—Douville, *Rec.*, 1907, 693.—Fiebiger, *Z. f. Tm.*, 1902, VI, 1.—Gheorghiard, *Ann.*, 1905, 153.—Marek, *Z. f. Tm.*, 1905, IX, 54.—Moussu, *Rec.*, 1897, 81, 558.—Petit, *Rec.*, 1902, 21.—Siedamgrotzky, *S. B.*, 1887, 71.—Sperling, *D. t. W.*, 1898, 270.

**Hematothorax.** The presence of pure blood in the chest cavity is called hematothorax, in the limited sense of the word. However, those cases are frequently included where a serous or purulent exudate or transudate are more strongly stained with blood. Such hemorrhages occur after intense infections, in exhausting diseases, in consequence of rupture of vessels in the thoracic cavity or after external traumatic injuries.

The symptoms simply point to an accumulation of fluid in the thorax. The detection of its hemorrhagic character is only possible by exploratory puncture. One may, in addition, observe the symptoms of an acute anemia, which correspond in degree with the amount of blood lost.

The treatment depends on the primary disease and the cause of the affection. Small amounts of blood may be completely absorbed.



# Diseases of the Digestive Organs

## SECTION I.

### DISEASES OF THE BUCCAL CAVITY.

1. **Catarrh of the Buccal Cavity. Stomatitis catarrhalis.**  
(*Stomatitis Simplex*, *Stomatitis Erythematosa*, *Maulcatarrh* [German].)

**Etiology.** Catarrh of the buccal mucosa may occur primarily after injury by sharp parts of plants, by pointed foreign bodies, such as fragments of bones, nails, etc., also in consequence of faulty teeth, improper bridle bits, by rough examination of the mouth. The buccal cavity of cattle may develop a catarrhal or even an ulcerative inflammation after penetration into the mucosa of licked up hairs or of the bearded grains of barley. (Koiranski.)

Scalding by hot feed (distillery slop, broth) or hot medicinal applications may also cause inflammations.

Chemical irritants may likewise be the causative factors, particularly such as are applied externally as medicines, and may accidentally be licked up by the animal or otherwise come in contact with the mucosa of the mouth. It sometimes occurs that animals drink of solutions of carbolic acid or corrosive sublimate, prepared and used for purposes of disinfection. Occasionally internal medicines, when used in too strong a concentration, may exert a caustic effect, and this is frequently the case with tartar emetic. Some medicines, like mercury, iodine or lead, may produce a catarrhal affection of the mouth after long-continued internal use. This condition may also be produced by strongly fermenting distillery slop, or other fermenting food materials. The milk of animals suffering from diseases of the udder, from foot-and-mouth disease, or from variola, produces inflammation in the mouth of the sucking young. Further causes of catarrhal inflammations are the poisons contained in such plants as spurge, hemlock, mustard, ranunculaceae, etc., occasionally also Swedish clover and fresh, insufficiently dried hay. Catarrhal inflammation of the mucous membrane of the mouth

of dogs has repeatedly been seen when these animals have been running around in fields abounding with stinging nettles. The hairs of the latter get into the mouth on inspiration and penetrate the mucous membrane. (Rohr.)

Feed infected with various fungi is believed to be particularly prone to cause inflammation. However, the experiments of v. Tubeuf have failed to demonstrate the correctness of this claim. (Fühlings, Landw. Zeitung, 1904, 467.)

Feed plants are subject to infection with the following rust and other fungi; the rape destroyer (*Polydesmus exitiosus*), the fungus of Swedish clover (*Uromyces occultus*), the rust of grain (*Puccinia graminis*), the rust of reed (*Puccinia arundinacea*, also mildew (*Erysiphe communis*) and *Tilletia caries*.

Caterpillars or the hairs of caterpillars mixed with feed cause inflammation both in consequence of traumatic injury and also on account of the presence of acrid poisons; injurious caterpillars are especially *Bombyx processionalis* and *Portheia chrysorrhœa*. Similar effects are produced by plant lice when present in large numbers; mites (*Acarus farinæ*?) are likewise said to produce such inflammatory processes.

**Secondary** catarrh of the buccal mucosa starting from the gums or their neighborhood may be due to the eruption or change of the teeth (gingivitis, oulitis).

Catarrh is frequently seen in the course of infectious and intestinal diseases, in which decomposition products of the saliva and the desquamated epithelia accumulating in the buccal cavity undoubtedly play an important rôle. Catarrh of the buccal mucosa is also common in inflammatory conditions of the neighboring organs (pharynx, larynx, salivary glands). A mucous membrane, debilitated by general nutritive disturbances, rachitis, anemia, marasmus, becomes predisposed to the development of catarrhal affections. This is also, partially at least, the reason why disease of the mucosa of the mouth is seen occasionally in dogs during gastro-intestinal inflammations or sepsis.

Finally, stomatitis occurs during the prodromal stage of specific infectious diseases (foot-and-mouth disease, infectious pustular stomatitis of horses, variola, rinderpest, malignant catarrhal fevers, scorbutus, diphtheria, etc.).

**Symptoms.** In acute stomatitis, an increased sensibility of the mucosa of the mouth is observed, and this causes the animal to take and chew its food in a more careful, deliberate manner. Thirst is generally increased. On examination, the mucous membrane exhibits an increase of temperature and congestion; the increased redness may be uniform or in patches; tenacious mucus is present. Sometimes there is some swelling, and in horses it is often considerable on the hard palate (frog on hard palate).



This is due to a marked hyperemia caused by compression of the venous plexus. Sometimes the tongue shows edematous swelling with transverse furrows or dental impressions. Occasionally, the excretory ducts of the mucous glands become occluded, and then small grayish nodules (stomatitis follicularis) are seen here and there, particularly on the internal surfaces of the lips. These may exceptionally form small, shallow ulcerations, which generally heal promptly; however, larger ulcers are sometimes found and such may also appear on the skin in the neighborhood of the lips and the nose; the lymphatic glands and their vessels may also become swollen (Kohnhäuser). Vegetable barbs which penetrate the mucosa also occasionally lead to the formation of small ulcers, up to pea size; the vegetable parts which have caused the ulceration may generally be seen at their base (Koiranski). In certain cases of stomatitis, an edematous swelling of the lips and cheeks, also a catarrhal inflammation of the nasal mucosa, may be observed.

The saliva is increased, salivation occurs, and a foamy fluid with large air bubbles adheres to the lips. Owing to the decomposition of the saliva and of the desquamated epithelia, a sweetish disagreeable smell becomes manifest (*foetor ex ore*).

The loosened epithelia, which are found on the surface of the tongue mixed with particles of food, form a light or dark grayish, occasionally greenish or brown cracked layer, which in carnivora hides the red flesh color of the adjacent muscular layer.

In cattle and in cats, the epithelial covering of the filiform papillæ, which is even normally well developed, proliferates a good deal and the upper surface of the tongue becomes markedly white, or yellowish and rough. A similar, though generally less marked, deposit may also be seen in other parts of the buccal cavity, such as the gums, the internal surface of the cheeks, the corners of the lips.

In fowls, the comparatively thick epithelial layer becomes dry and opaque and forms a pseudomembranous covering (*pellucula linguæ, pituitas*).

**Chronic stomatitis** rarely leads to disturbances of the general health, which are then similar to those of the acute form, but usually milder.

**Course.** Primary stomatitis always takes a favorable course. After the swelling and the hyperemia subside, the ulcerations which may have been formed, heal with the development of a new epithelial covering. Stomatitis follicularis likewise ends in recovery in one to two weeks, even if neighboring cutaneous parts are involved, although scars may remain if the ulceration has been deep. The course of secondary stomatitis depends upon the nature of the underlying disease.



Most veterinary authorities describe this disease under the name of stomatitis aphthosa. The authors believe that this is not quite correct because it is inadmissible to designate vesicles as aphthæ. The latter term more properly designates pseudomembranes of the buccal mucosa (see page 188). In distinguishing this affection from foot-and-mouth disease (aphthæ epizooticæ) it is frequently designated as aphthæ sporadicæ.

**Occurrence.** The disease has, so far, been observed only in horses and cattle. It generally occurs sporadically (Guittard, Iwersen, Vontobel, Pr. Mil. Vb.), but among horses also enzootically (Dieckerhoff, Bochberg) or even epizootically (Theiler). Epizootics of the disease have been observed particularly in South Africa.

**Etiology.** Pasturing in rape stubble fields or ingesting much Swedish clover have frequently been held to be the cause of the disease, which was then believed to be due to mycotic invasion (*Polydesmus exitiosus*, *Uromyces occultus*). (See also Clover disease.)

Michaelis observed three cases where the disease came on simultaneously with an exanthematous eruption near the feet after feeding on potatoes which had already germinated. Bochberg saw five horses of one owner affected after they had received mouldy and partly dusty feed. Dieckerhoff saw 50 horses out of 100 of the Trakehn Stud become sick successively, and therefore believed that the disease was infectious and that it found its portal of entrance through a lesion of the buccal mucosa. However, since Dieckerhoff's and also Bochberg's inoculation experiments were not successful, they deny the infectious nature of the disease. Still, Theiler saw in South Africa an infectious vesicular eruption of the buccal mucosa of horses ("Blauwtong" the blue-tongue of the Boers) which could be transferred by inoculation, but which does not spread rapidly by natural infection.

**Symptoms.** In horses the buccal mucosa becomes reddened, sensitive and hot and there may occur a slight elevation of temperature, at the same time vesicles appear of the size of a millet seed, lentil or bean, filled with a clear watery or yellowish serous fluid, thin-walled, flattened or even umbilicated. These vesicles are seen on the inner surfaces of the lips, on either side of the lingual ligament, on the gums between the teeth, in the neighborhood of the angles of the mouth, on the lips and on the sides of the tongue. The vesicles burst in three to four days, leaving superficial erosions which remain partly covered for some time by the whitish fragments of the torn wall of the vesicle. Adjoining erosions may become confluent and form irregular defects in the epithelial covering. Vesicular stomatitis occurring in South Africa at this stage exhibits a dirty bluish discolored tongue (hence the name of the disease). The intensely red base of the erosions soon fills up and a new epithelial covering is formed in five to six days, when all traces of inflamma-

Aphthæ in veterinary medicine designate vesicles of the buccal mucosa which are generally filled with a serous fluid, while in human medicine, according to the histologic investigations of Bohn, Sechech and Fraenkel, aphthæ are pseudomembranes formed by desquamated fibrin, below which the tissues are comparatively intact. For the sake of clearness and from comparative pathologic considerations this term which is generally used in human medicine is retained, though histologic studies on the part of veterinarians have not come to the knowledge of the authors.

Out of a large group of diseases evidently quite variable as to their etiology, only two groups will be described in detail in the following chapters.

#### (a) Aphthous Inflammation of the Mouth of the Young.

(Socalled *Maulschwaemmchen*, *Kahn*, or *Soor der Saeuglinge* [German]; *Stomatite ulcéreuse des agneaux et des chevreaux* [French].)

This contagious disease of sucklings is characterized by the development of pseudomembranous deposits which are, at first, whitish, later grayish or grayish yellow, on a more or less inflamed buccal mucosa.

**Occurrence.** The disease usually occurs in the form of an epizootic. Lambs are most frequently affected, more rarely kittens, also sucking calves and foals. The animals generally sicken during lactation, more rarely directly after weaning and when being fed with flour pastes.

Koenig (1855) and Spinola (1858) mention affections under the name of scorbutus of lambs (Gips) or of symptomatic aphthous disease of lambs which probably belong to this group. The same is true of the disease called croupous stomatitis of sucking foals (Hartmann) and of the ulcerative stomatitis of lambs.

**Etiology.** The epizootic character of the disease suggested its infectious nature, and indeed Delafond succeeded in transferring it artificially. From the pseudomembranes and from the internal organs of sick animals, Besnoit isolated large Gram-positive cocci, arranged in groups of two or in chains of four; cultures of these, when inoculated, produced the disease in lambs and kittens. Whether the pseudomembranes are primarily due to the bacillus necrophorus, the disease therefore representing a true necrobacillosis as indicated by Mohler, has not been proven. However, in its severest form the disease is very similar to necrobacillosis of the buccal mucosa (infection with *b. necrophorus*).

Zürn claims that the disease is due to *oidium albicans*. In consequence of this assertion the disease has generally been called "Soor" ("Maulschwaemmchen," Kahn), though the soor or thrush mould has never been identified definitely in this disease.



**Diagnosis.** The white or yellowish spots which are formed together with the symptoms of stomatitis and the vesicles on the margins of the lips furnish a very characteristic picture. The presence of vesicles on the margins of the lips, the inflammatory halo around the lips, and the shallow ulcerations distinguish the disease from thrush (Soor), while the absence of vesicles on the mucosa differentiates it from vesicular stomatitis. In severe cases of aphthous stomatitis, ulcerative stomatitis may be thought of; but as such severe cases usually occur side by side with mild cases, the true nature of the disease can readily be recognized.

**Treatment.** In consideration of the very contagious character of the disease, healthy young animals and their mothers must be separated from the sick lambs. The healthy animals should be kept under observation and all new cases of infection are to be separated promptly. The pens must be subjected to a thorough cleaning and disinfection. Since the sick animals can, in consequence of the painful character of the disease, not easily suck or not at all, they should be fed with milk obtained from their mothers by milking; or if they are old enough to be weaned they may receive some other suitable liquid food.

The local treatment consists in frequent washing of the mouth with disinfectant fluid, for instance, 4% boracic acid solution, 3-4% solution of chlorate of potash, 1-2% solution of sulphate of copper. These solutions should be introduced into the buccal cavity with a syringe or an irrigator.

**Literature.** Besnoit, Rev. vét., 1901, 213.—Fränkel, V. A., 1888, CXIII, 484.—Gips, A. f. Tk., 1885, XI, 296; T. R., 1888, 209.—Hartmann, Ö. Vj., 1880, LIII, 5.—König, Pr. Mt., 1855, 56-109; 1872-73, 126.—Zürn, Pfl. Parasiten, 1874, 1883.

#### (b) Infectious Papulous Inflammation of the Mouth of Cattle. *Stomatitis papulosa.*

(*Gutartige Maulseuche* [German] [OSTERTAG & BUGGE]; *Stomatitis follicularis* [HESS]; *Stomatitis papillaris* [DEGIVE].)

Contagious papulous inflammation of the mouth of cattle is an acute specific disease caused by an ultramicroscopic micro-organism; it is afebrile and is characterized by the appearance of flat papules on the buccal mucosa and often also on the muzzle.

Since the papules on the buccal mucosa have a certain similarity in their structure with the aphthæ of man, it is proper to consider the disease in this place.

**Historical.** The etiology of the disease which made its appearance, epizootically, among Bavarian steers in the stockyards of Berlin (Tiarks) has been cleared up by Ostertag & Bugge (1905), who also showed that



it was identical with a disease described by Hess in 1899, under the name stomatitis follicularis, and by Degive (1884) as stomatitis papillaris. The sporadic aphthous disease of Deppe (1899) and the infectious stomatitis seen by Haag (1907) in Bavaria likewise appear to be identical with this disease. According to Tiarks (1904), it is common in the Prussian provinces of Posenia and East Prussia. Finally, some of the diseases called pseudo foot-and-mouth disease of cattle (*q. v.*), probably are also identical with this affection.

**Etiology.** The cause of the disease is an ultramicroscopic microorganism which is found in the pathologic lesions and in the blood of the sick animals.

**Pathogenicity.** When particles of the pathologically changed mucosa are inoculated into the buccal mucosa of healthy cattle, they produce the disease in its typical form; it can also be produced by subcutaneous and intravenous injections of the blood of sick animals. Filtered blood serum also caused the disease in the experimental animal in one case, but it was not effective in subsequent transfer experiments, although the defibrinated blood of the same animal remained virulent. If the pathologic products are transferred to the nasal, ocular or vaginal mucosa, the disease is not produced. Spontaneous transmission to healthy calves has also been observed. Younger animals are more susceptible than older ones.

How natural infection is produced has not yet been shown. The experiments of Ostertag & Bugge make it possible to believe in an inoculation with the food through injuries in the buccal mucosa.

**Pathogenesis.** The blood vessels are dilated in circumscribed places of the buccal mucosa; this is followed by migration of leucocytes into the lymph spaces in the neighborhood of the blood vessels of the superficial layers of the mucosa, and between the epithelial cells of the stratum germinativum; in consequence of this, the epithelial covering and the papillæ of the mucosa become thickened. In the meantime the epithelial cells become vacuolated; this vacuolation increases toward the surface and a desquamation of epithelia occurs, so that the papillæ remain covered only by the deepest epithelial layers. In the further course of the process, the epithelia covering the elongated papillæ proliferate and form a new covering. If the upper portion of the papillæ has been destroyed, the latter suffer a diminution in size.

**Symptoms.** The incubation stage after artificial infection lasts an average of two weeks, although transitory, but not characteristic, symptoms may sometimes appear after seven to eight days. Flat nodules of from millet seed to lentil size appear on the internal surfaces and on the margins of the lips,



also on the hard palate, particularly on its sharp margin, at the corners of the mouth, and on the inferior surface of the tongue, on the internal surface of the cheeks, and also exceptionally on the soft palate. These nodules may become confluent in the further course of development and form larger patches. Similar nodules often are formed in the integument of the muzzle. The nodules and the area surrounding them at first appear intensely reddened, later on they assume a yellowish-gray color and are then surrounded by a red area. They are firmly connected with the underlying surface and cannot be removed easily, whether as pseudomembranes or otherwise. In the further course the epithelial covering desquamates without the preliminary formation of vesicles and central circular depressions are formed, with a granular, frequently excavated, base, which may either show an intensely red or a blackish color, depending upon the pale red or the grayish-black color of the mucosa of the sick animal. While the erosion grows to the size of a bean or even to that of a silver dollar, the base becomes yellowish. After eight days the erosions are closed by a new epithelial covering, but the healed spots may be recognized for as long as one month by their intensely red color and their roughened surface. Recurrences occur frequently and repeatedly, so that the course of the disease may extend over months; however, it always ends in recovery. Other symptoms are usually absent, although in more severe cases there may be a formation of crusts on the skin, general emaciation, and a bad smell from the mouth.

Liénaux noticed in a cow salivation, grating of teeth and diarrhea in addition to the symptoms above described. Post-mortem examination showed erosions and loss of substance along the whole gastro-intestinal tract similar to those in the mouth. Artificial inoculation was not successful in contradistinction to what occurs in contagious papulous inflammation. Hess mentions the occurrence of very small vesicles in the center of nodules which burst soon and attain the size of a millet seed to that of a pea.

**Diagnosis.** The presence of the peculiar flat, nodule-like formations on the buccal mucosa, and occasionally on the muzzle, without disturbance of the general health and without involvement of the feet, the slight extension of the lesions, their eaten-out appearance, with only superficial loss of substance sufficiently characterize the disease. It may be distinguished from vesicular stomatitis by the absence of vesicles in the mouth, and by the presence of the characteristic nodules in the neighborhood of the existing erosions; besides there is increased secretion of saliva in vesicular stomatitis. The same differential features also distinguish the affection from foot-and-mouth disease in which large vesicles are often seen on the back of the tongue or on the feet. In aphthous stomatitis of sucklings, pseudomembranous deposits are developed on the usually reddened tongue and the disease affects only sucklings and young animals which have just been weaned.

**Treatment.** This is similar to that employed in vesicular stomatitis. Considering the contagious nature of the disease, sick animals should be separated from the healthy ones.

**Literature.** Degive, Ann., 1884, 369.—Deppe, A. f. Tk., 1899, XXV, 199.—Haag, W. f. Tk., 1907, 906.—Hess, Kongr. Bern., 1899, 382.—Ostertag & Bugge, Z. f. Infkr., 1905, I, 3.—Tiarks, Pr. Vb., 1904, II, 72. (Compare also the literature on Pseudostomatitis; Pseudomaulseuche.)

**Other Diseases Similar to Stomatitis.** Aside from aphthous stomatitis of sucklings and from contagious papulous stomatitis, diseases have been observed in cattle and exceptionally in sheep which, on the basis of pathologic-anatomical changes, are best classified with aphthous stomatitis. Among them are to be mentioned the following: sporadic stomatitis (Maulseuche, Utz), affections similar to foot-and-mouth disease (Bang, Stribolt, Andersen, Kern), pseudo mouth-disease or stomatitis erosiva (Pusch), thrush of cattle (Hajual), epizootic inflammation of the mouth of sheep (Vigadi), stomatitis ulcerosa in cattle (Bedel), pseudo foot-and-mouth disease (Kantorowicz), stomatitis mycetica (Mohler), stomatitis pseudothosa (Cadéac).



Fig. 30. Pseudo-Aphthous Inflammation in Cattle (According to Kern).

Concerning their etiology, these affections may be divided into three groups. The first group comprises those diseases which are contagious and which consequently must be due to an infectious agency, which in part of these cases may be the bacillus necrophorus (see Necrobacillosis). In the affections of the second group (Utz, Bruemmer, Bang, Stribolt, Andersen, Kantorowicz) injurious food appears to be the exciting cause. The appearance of such diseases has followed upon feeding with green clover. They are not contagious. In the other non-contagious diseases of this kind, nothing positive is known as to their cause. Pusch believes that they may be due to some noxious agent which becomes a disease producer only when the resistance of the organism has been lowered by overexertion (as in railroad transit), irregular care or



sudden change in feed. Fungi and moulds have been accused as the causative factors, but nothing has been proven in this respect.

It is not impossible that some of the forms here enumerated belong to the contagious papulous form of stomatitis (see page 190).

As to the **clinical picture**, the aphthouslike diseases may, for convenience sake, be divided into two groups. In one group the buccal mucosa only or possibly also the integument of the muzzle is involved. The mucosa of the hard palate, the gums, more rarely the sides and the tip of the tongue, the inner surface of the lips and of the cheeks, present grayish-white to grayish-brown pseudomembranous deposits on a more or less reddened surface, or in other cases, round, intensely red erosions up to the size of a silver quarter, the base of which is partly covered with a grayish-red, or yellowish, thin, flaky or thicker deposit. In some cases one also sees numerous pinhead hemorrhages in the papillæ of the mucosa. In some animals the muzzle shows lenticular prominences and erosions of the epithelial covering (Fig. 30). Only exceptionally does the disease assume a febrile course and lead to salivation and disturbances in the ingestion of food.

The clinical picture of the second group is characterized by the simultaneous affection of the buccal mucosa, of the integument of the muzzle, of the integument of the extremities, and of the udder. With an elevation of temperature, the appetite is diminished or entirely lacking. Profuse salivation appears and smacking noises are heard just as in foot-and-mouth disease. The mucosa of the mouth is intensely reddened and covered with corrugated, diffuse, pseudomembranous deposits, especially on the hard palate, the gums, the inner surfaces of the lips. The pseudomembranes come off in shreds and the erosions heal in a few days. Lenticular yellowish-red or grayish nodules or crusts with a central depression appear on the muzzle and exceptionally on the nasal mucosa. Similar nodules sometimes appear on the mucosa of the mouth. The lower extremities show evidences of a dermatitis which is similar to that seen in clover diseases (see under this heading); on the skin of the udder, nodules of the size of a lentil with several vesicles and subsequent crust formation were seen by Kantorowicz.

The disease always takes a favorable course either within a week, or in the variety last described within two to three weeks, slight emaciation having become manifest in the meanwhile.

The **differential diagnosis** has to consider foot-and-mouth disease. It is distinguished by the simultaneous vesicular eruption at the ends of the extremities and on the buccal mucosa, the involvement of the back of the tongue and the absence of the peculiar pseudomembranes on the mucous membranes of the mouth. In the forms of stomatitis here described, the contagious character is usually absent, also the involvement of the extremities and of the udder.

**Treatment** becomes necessary only in the more severe forms with dermatitis at the extremities. The principles of the treatment are the same as those in catarrhal stomatitis (see page 185). Most cases end in recovery without any treatment at all.

**Literature.** Andersen, *Maanedsskr.*, 1901, XIII, 182.—Bang, *Maanedsskr.*, 1899, XI, 157.—Bedel, *Bull.*, 1904, 545.—Cadéac, *J. vét.*, 1906, 556.—Hajnal, *Vet.*, 1900, 71.—Kantorowicz, *Z. f. Infkr.*, 1906, II, 559.—Kern, *A. L.*, 1907, 387.—Mohler, *Rec.*, 1905, 112.—Pusch, *D. t. W.*, 1906, 133.—Stribolt, *Maanedsskr.*, 1901, XIII, 130.—Utz, *B. Mt.*, 1890, XXVI, 135.—Vigadi, *A. L.*, 1905, 345; 1906, 423.

#### 4. Thrush. *Stomatitis oidica*.

(*Soorkrankheit, Schwaemmchen* [German]; *Muguet* [French].)

The term thrush designates patches formed on the mucous membrane of the mouth and pharynx by the mycelium of *oidium albicans* or *monilia candida*.

**Historical.** The disease has heretofore only been found positively in fowls, especially in pigeons (Eberth, Martin). Its occurrence in other animals is at present doubtful. (Gravitz has produced it experimentally in a dog.) Zürn described under the same name a disease of foals and calves, but he has not demonstrated the typical fungus and his description of spots and vesicles suggests that he was dealing with a more severe form of stomatitis vesiculosa or stomatitis aphthosa. Hajnal observed a contagious disease in young cattle which he believed to be thrush, but he did not demonstrate the fungus (see page 193). The fungus of thrush was discovered in 1840 by Berg and Gruby, and studied more minutely by Gravitz, and particularly by Plaut (1887). The latter author demonstrated the identity of *oidium albicans* and *monilia candida* and the transmissibility of thrush in fowls. (It is also found in man, almost exclusively in infants or young children. [Translator].)

**Etiology.** *Oidium albicans* (*saccharomyces albicans*, *monilia candida*) is probably only a stage in the development of one of the higher hyphomycetes (Plaut) with multiple-branched filaments which possess rounded ends, while the spores are oval, elongated and sharply circumscribed (Fig. 31). On solid media the colonies consist of roundish cells only, while in fluid media they become elongated into extensive filaments. The organism grows well on decaying wood, on fresh cowdung and in curdled, but not in fresh, milk (Plaut). It invades particularly the mouth of debilitated and young animals, and the sojourn in damp, dark, poorly ventilated places favors the appearance of the infection.



Fig. 31. *Oidium albicans*. From the mycetically degenerated eye of an inoculated rabbit; hyphae, spores and a number of leucocytes in groups. (From Plaut.)



Martin and Klee claim to have observed the transmission of thrush from sick cattle to chickens and to one turkey.

**Symptoms.** The characteristics of thrush are grayish-white or yellowish, later on brownish points, spots or even larger pseudomembranes adhering to the underlying tissue, which does, however, not show any signs of inflammation. During the course of the disease, the most superficial epithelial layer perishes and the colonies of fungi now are seen on the surface. However, this occurrence does not loosen the intimate connection of the fungi with the tissues. Similar pseudomembranes are found in the pharynx and in the crop; when this occurs, the birds are listless and perish in spite of good appetite. (See catarrh of the crop.)

\* Under the microscope one sees in the scraped off masses, besides desquamated epithelia, some round cells and numerous curved and straight filaments and between the latter spores.

**Diagnosis.** Thrush is distinguished from aphthous and other inflammatory forms of stomatitis by the absence of an inflammatory reaction in the neighborhood of the lesions. It is necessary to demonstrate the typical fungi microscopically before an absolute diagnosis can be made.

**Treatment.** The mouth must be kept clean and it may be painted with a 2.5% solution of borax, or, according to Klee, with a solution of bichloride of mercury (1:1000). If the latter solution is employed, great care is required. If the crop is involved, it should be irrigated with a 2% boracic acid solution. (See catarrh of the crop.)

The places where the fowls are kept should be washed with bisulphate of calcium; the feeding vessels should be disinfected by scalding. In this manner a spread of the disease may be prevented.

**Literature.** Klee, Geflügelkrkh., 1905, 81.—Martin, M. Jb., 1882-83, 125.—Voigt, Jb., f. p. M., 1903, 1, 575 (Lit.).—Zürn, Pfl. Parasiten, 1874, 188; Geflügelkrkh., 1882, 130.

## 5. Phlegmonous Inflammation of the Mouth. Stomatitis phlegmonosa.

(*Stomatitis erysipelatos*a, *S. erosiva*.)

**Etiology.** After the ingestion of large masses of food contaminated with rust and other fungi, a severe inflammation of the buccal mucosa is occasionally seen in horses and exceptionally in sheep, complicated by suppuration and necrosis of the submucous connective tissue. De Hahn & Hoogkammer saw in East Indian horses an inflammation of the skin of the



face, complicated with erysipelatous stomatitis, caused by fungi (*hyphomycosis destruens equi*). (See Vol. I.) Food which contains many caterpillars or their hairs (*porthesia chrysorrhea*, *bombyx processionalis*) may act likewise. Lüthens and Köster have shown experimentally that otherwise good food, but mixed with procession caterpillars or their hairs spread on oak leaves, was able to produce a severe stomatitis in horses.

Irritants, such as lye, acids, carbolic acid, croton oil, and scalding of the mouth, are likewise able to produce severe stomatitis. Traumatic lesions of the mucosa may give rise to inflammatory processes of the deeper tissues.

The disease may appear secondarily after purulent or gangrenous processes of neighboring organs (purulent inflammation of the entrance of the esophagus) or in connection with infectious diseases, such as strangles, cattle plague, anthrax, hog cholera, morbus maculosus (*purpura hemorrhagica*), malignant catarrhal fever, diphtheria of fowl or calves, dog typhus. Erysipelatous inflammation of the tongue forms part of the picture of the disease known as blue-tongue of sheep in South Africa. (See Vol. I.)

**Symptoms.** The disease sets in with distinctly inflammatory phenomena, great tenderness, intense reddening and swelling of the mucosa; these are generally accompanied by an elevation of temperature. The swelling later on increases still more, and external inspection shows particularly swelling of the lips and of the neighboring portions of the cheeks. The swollen lower lip has fallen and a profuse saliva drips in long threads from the corners of the mouth. The mucous membrane on the inner side of the lips and cheeks is very tense, bluish-red, and forms thick folds back of the teeth, on the floor of the buccal cavity, but especially on both sides of the lingual ligament. The swelling of the tongue is sometimes so extensive that its tip protrudes between the incisors and the organ shows on its lateral margins the impressions of the molars, and on the tip the impressions of the incisors; it is, at the same time, tense and hard.

In the further course, symptoms of pharyngitis make their appearance, and interference with the ingestion of food and water may become complete. The nasal mucosa may also become involved and then a mucous yellowish-brown nasal discharge appears. The submaxillary and the peripharyngeal lymphatics become swollen, hard and painful.

At this stage, the disease has, in most cases, reached its height and recovery sets in slowly. In the severe cases, however, pale yellowish, mushy deposits and pustules appear on the swollen mucosa up to the size of a pea (observed by Bertsche in sheep) and these again lead to the formation of irregular, angry-looking ulcers. Occasionally abscess formation occurs with ichorous, dirty, ill-smelling pus, mixed with frag-

ments of necrotic tissue and parts of food stuffs. In these cases there is fever, listlessness, and indican appears in the urine.

**Course and Prognosis.** The primary form of phlegmonous stomatitis generally takes a favorable course. If there is no necrosis of tissue and no abscess formation, complete recovery usually takes place within two or three weeks; even in the severest cases recovery may occur under the proper surgical treatment. Exceptionally, however, the inflammatory process spreads to the larynx, the posterior wall of the pharynx and along the trachea, which then usually leads to an unfavorable termination. The latter also occurs in secondary stomatitis, especially if it occurs in connection with anthrax, hemorrhagic septicemia or with other malignant affections.

**Treatment.** If the inflammation has been caused by noxious food, a change of the latter and of pasture must be instituted. Besides, irrigations and mouth washes, as generally used in stomatitis, are indicated; purulent and ichorous inflammation requires proper surgical interference.

**Literature.** Berndt, A. f. Tk., 1887, XIII, 364.—Bertsche, B. Mt., 1890, 137.—Klimmer, Veterinärhygiene, 1908, 204, 230 (Lit.).—Köster, Pr. Mil. Vb., 1889, 84.—Zörn, Pfl. Parasiten, 1874, 187.

**Stomatitis Gangraenosa Agnorum.** Gangrenous Stomatitis of Lambs. Riotta repeatedly has observed a severe epizootic disease of young lambs characterized by the appearance of vesicles up to the size of a copper cent, with thickened margins. Most of the sick animals die within a few days, and the post-mortem examination, aside from the lesions of the mouth, always showed tubercle-like foci in the liver. In these foci was found the bacterium subtilis agnorum which produced ulcers on the mucosa on being inoculated into the buccal cavity of healthy animals. (Rivolta, Giorn. di Anat. fisiol. e pathol. 1883-78.)

## 6. Ulcerative Inflammation of the Buccal Cavity. Stomatitis ulcerosa.

(*Mundfaeule* [German]; *Stomacace*.)

Ulcerative stomatitis is especially a disease of carnivora, consisting in an acute inflammation of the gums, which soon leads to necrosis and ulcerative destruction.

Severe forms of stomatitis of herbivora caused by spoiled food, including aphthous stomatitis, are included in this affection by some authors (Friedberger & Fröhner, Gips, Cadéac). But their clinical picture is so different from that of stomatitis ulcerosa, that such a classification does not appear justified.

**Etiology.** The disease generally occurs among high bred pet dogs and cats, especially among weakly and ane-

animals, after distemper or in the course of rachitis, etc. Older dogs with tartar and caries of the teeth are affected most frequently, particularly if they have been nourished insufficiently.

The direct causes of the inflammation and of the subsequent tissue necrosis are not known. The character of the morbid process points to an infection as does likewise the experience that a similar disease in man frequently assumes an epidemic character. Tartar on the teeth predisposes to infection in consequence of the loosening of the gums; anemia and cachexia act by diminishing the resistance of the tissues.

It is possible that pathogenic bacteria which are normally found in the saliva may have part in the causation of the disease. Fiocca has demonstrated a bacillus *salivarius* septicus and the staphylococcus pyogenes aureus in the saliva of dogs and the bacillus coli communis in the mouth of sucking cats. One also must think of the presence of the bacillus necrophorus. However, inoculation of material from sick into healthy animals has not been successful in transferring this disease (Cadéac).

Exceptionally a stomatitis which is primarily benign may pass over into an ulcerative inflammation in consequence of secondary specific infection. The disease also appears as a part of the clinical picture of the infrequent scorbutus in dogs and swine and of chronic hog erysipelas. (See Vol. I.)

The disease is similar in its symptoms to inflammation of the mouth seen in connection with mercurial poisoning after repeated inunctions with gray ointment or after excessive injections of corrosive sublimate (stomatitis mercurialis). (According to Cadéac's experimental investigations, the real cause of this affection is claimed to be the bacillus necrophorus.) Sometimes other agencies like lead, copper or phosphorus may have a similar effect.

**Symptoms.** The inflammation begins almost without exception at the margins of the gums, around the neck of the incisors of canines, especially in the spaces between the individual teeth. The gums of the molars usually become affected later. The gums at the affected places appear dark or sometimes bluish-red, swollen, very painful, and on being touched bleed but slightly. After one or two days, the free margins of the gums become changed into a pale yellowish or dirty-greenish smeary, mushy mass, which can easily be removed; the live tissue in their neighborhood looks bluish-red and swollen. After the expulsion of the mushy masses, one sees, around the neck of the teeth, ulcers with margins and bases covered by necrotic, discolored detritus. The erstwhile round ulcers become confluent forming an ulcerated discolored surface; the necks of the teeth are left uncovered. After the process has spread into the alveoli, the teeth become loosened and can be extracted easily, or they may fall out spontaneously.

In the further course similar ulcers are formed on the mucosa of the lips and cheeks; these ulcers are found opposite the



primary ones on the gums and they are probably due to contact infection. The lips and the cheeks become swollen. In very severe cases the inflammation may extend into the maxillæ and cause necrosis of portions of the bone.

Sometimes, however, the process may begin on the lips or at the angle of the lips and may extend toward the neighboring portions of the integument (Noma) while the gums remain intact. (Friedberger & Fröhner.)

There is always an extremely disagreeable, repulsive, sweetish, cadaverous smell present, emanating from the buccal cavity (*foetor ex ore*), which pervades the whole space in which the sick animal is kept. There are also symptoms of acute catarrh of the mouth, especially profuse salivation. The saliva is fetid, mixed with necrotic shreds and also with blood. The submaxillary and the cervical glands are acutely swollen and the inflammation extends also to the salivary glands. The increased tenderness disturbs mastication, and the animals either take only fluid food or they swallow soft pieces of meat without chewing.

Later on the symptoms of septic infection develop, such as febrile elevation of temperature, rapid small pulse, diarrhea, prostration; the rapidly emaciating animals soon succumb.

**Course.** The disease takes a favorable course under favorable hygienic conditions, with the proper treatment and if the patients are still in good condition of nutrition. The inflammatory and necrotic processes become limited, the necrotic portions are shed and healthy granulations form from the margins and from the base of the ulcers. The disease ends then in complete recovery within one to two weeks. If the cases are neglected, the animals become much emaciated and a general infection or exceptionally an aspiration pneumonia with pulmonary gangrene develops. (Müller has observed this course in a dog, the authors a similar case in a lion.)

**Diagnosis.** Since ulcerating stomatitis may form an accompanying symptom of scurvy, the other symptoms of this disease must be looked for; the absence of hemorrhage indicates that the stomatitis occurs independently. In young pigs chronic erysipelas must be excluded; only the history and the accompanying conditions can usually give information as to whether the ulceration is due to poisoning.

**Treatment.** Next to washing and irrigation of the buccal cavity with solutions already mentioned (page 185), careful cauterization of the ulcers with nitrate of silver is indicated. Crusts form on the cauterized places and, after the shedding of the former, clean granulating surfaces appear. Painting with the following solutions also gives favorable results: 1-2% solution of nitrate of silver, 2-3% solution of

chloride of zinc, pure turpentine, 5-10% solution of tannic acid, glycerine, 3% solution of creolin; deodorizing solutions may be used in addition, such as 0.3% solution of potassium permanganate. After the ulcers have become clean, mild disinfectant and astringent solutions are indicated.

During treatment, the animals have to be fed suitably with liquid, juicy nourishment (milk, meat). The teeth which have already become loosened must be removed, a procedure which in the beginning of the disease occasionally leads to a rapid termination of the pathologic process.

**Literature.** Cadéac, J. vét., 1907, 484.—Hébrant, Ann., 1903, 11.

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#### **Other Diphtheritic Inflammations of the Mucosa of the Mouth.**

Aside from hog cholera and diphtheria of calves and older cattle, other distinct diphtheritic inflammations occur occasionally on the mucosa of the mouth. Bang and Lauritsen saw diphtheritic pseudomembranes in hogs, (Lauritsen in very young sucking pigs), on the mucosa of the lips and the buccal cavity in connection with a painful swelling of the skin in the neighborhood of the snout, occasionally with diphtheritic necrosis of the mucosa of the septum of the nose. This was associated with very rapid emaciation. Stallmann saw a similar disease in goats with salivation, disturbed deglutition, lack of appetite, and high fever; most of the animals died. In the case of young pigs, curetting of the necrotic tissue followed by astringent irrigations gave good results. (Lauritsen, Maanedsskr. 1903, XV. 121.—Stallmann, Pr. Mt. 1880, VI. 51. See also Necrobacillosis.)

## SECTION II.

### DISEASES OF THE SALIVARY GLANDS.

#### 1. Salivation. Ptyalism.

(*Speichelfluss* [German]; *Salivatio*, *Sialorrhoe*.)

**Etiology.** Salivation is almost without exception a purely secondary symptom, seen most frequently in inflammation of the mucosa of the mouth and pharynx and due to a reflex irritation of the nerves which regulate the activity of the salivary glands. It is also a frequent, though not constant, symptom of parenchymatous inflammation of the salivary glands. Certain chemicals, especially mercury, pilocarpine, arecoline, nux vomica, more rarely arsenic, iodine, lead and copper likewise increase the secretion of saliva; also acrid substances and food contaminated with rust and other fungi. Disturbances of deglutition also are accompanied by salivation; this is especially seen in pharyngitis, in spasm and paralysis of the larynx, and in diseases of the esophagus. Diseases of the gastric mucosa (ulcer, catarrh, inflammation) may cause salivation by reflex irritation. Whether certain diseases of the sexual organs stand in a causal relation to salivation is a point not yet settled in veterinary literature. Anacker reports the interesting observation that a horse always showed salivation when taken to the knacker's yard.

**Symptoms.** Saliva flows abundantly out of the mouth, particularly at the corners, either as foam or in long strings, and contaminates the food and other nearby objects. Long-continued salivation finally leads to emaciation. Aside from these symptoms, the primary cause can generally be recognized.

**Treatment.** Apparently, primary cases of salivation may be treated by the subcutaneous injection of atropine (for large animals, 0.05-0.1 gm.; for small ones, 0.005-0.02 gm.) or scopolamine (0.01 or 0.002 gm.). This leads, however, usually only to temporary relief. In a case of Dieckerhoff's, continued treatment with arsenic (horses, 0.05-0.8 gm.) led to permanent improvement, while Zündel was successful in the treatment of a cow with iodide of potash. In most cases, the treatment must



be directed against the underlying primary condition and no direct treatment of the salivation is required. Diem obtained recovery in a case due to swelling of the parotid gland by repeated injections of pilocarpine, followed by inunctions with an iodine-iodide of potash ointment.

## 2. Inflammation of the Parotid Gland. Parotitis.

**Etiology.** Parotitis occurs, as a primary affection, with comparative frequency among domestic animals, after traumatic injury of the parotid region. It occurs rarely in epizootic form as a specific infection among cattle, horses, dogs, cats and goats. The cause of this form of the disease is probably an infective agent gaining entrance into the glandular tissue either through the salivary duct or through the general blood circulation. The analogous disease of man known as mumps or parotitis epidemica is beyond doubt due to an infection; according to Korsutschewsky, its cause is a micrococcus.

Bissauge has repeatedly seen parotitis as an epizootic among cattle in the neighborhood of Orléans in France and always simultaneously with mumps among children. In a dog a case occurred likewise during a mumps epidemic among children, and the affection spread to another dog. A diplococcus was obtained in pure culture from Stenon's duct (Busquet & Boudeaud). The infection of a dog from sick children was demonstrated beyond doubt by Prietsch.

Aruch saw inflammation of the parotid and of the submaxillary glands on one side among seventy horses, within one year and a half; as a rule there was suppuration. He repeatedly found beards of oats and of *Hordeum sylvaticum* in Stenon's duct and in the pus; in one case 100 beards were in Stenon's duct. Sometimes the inflammation is caused by *Brucella abortus* (*Brucella abortus*) (*Brucella abortus*) (Renault, Meyer) or by *Vicia tenuifolia* (Labat) and it is probable that these plants simply play a rôle as carriers of pathogenic bacteria.

Parotitis is frequently secondary in nature, particularly after inflammation of the pharynx. In this case the process probably extends along the connective tissues, but the inflammation may also spread from the pharynx along Stenon's duct. Salivary calculi may also cause inflammation in this long duct with secondary involvement of the glandular tissue.

As a part of the clinical picture in infectious diseases, parotitis occurs in strangles in the horse, also as a metastatic process in dog distemper and in influenza of the horse.

Chronic parotitis develops after repeated traumatic injuries (particularly in race horses) in the presence of salivary calculi, and in cattle in connection with actinomycosis.

**Anatomical Changes.** Acute parotitis leads to swelling and intense reddening of the glandular tissue and to interstitial serous infiltration of the interlobular connective tissue (paro-

titis parenchymatosa); at other times it leads to the formation of small purulent foci between the lobules; these may subsequently become confluent and form larger abscesses (parotitis apostematosa s. abscedens). Chronic inflammation leads to an increase of the interlobular connective tissue, partial atrophy of the lobuli and induration of the whole gland (parotitis chronica indurativa).

**Symptoms.** **Acute inflammation** exhibits primarily a diffuse or on the contrary a more circumscribed swelling of the parotid region, which may extend into the neighborhood as a collateral edema. The edematous infiltration in the neighborhood leads, especially in cattle, to laryngeal stenosis and disturbances of deglutition. The gland itself is painful, and as in inflammation of the throat, the head is stretched, or in unilateral affections of the gland, held obliquely. The animals chew with care, and they do not ingest large morsels, because every depression of the lower jaw compresses the tender parotid gland. The secretion of saliva is usually increased (Müller), but it may also remain normal (Friedberger & Fröhner). Sometimes the disease is preceded by symptoms of stomatitis and pharyngitis, complicated with immobilization of the swollen tongue and intense swelling of the gums (Aruch).

Purulent inflammation always leads to inflammatory edema of the neighboring connective tissue, consequently the individual lobules cannot be distinguished; the swelling is usually of a high degree and continues down toward the neck and into the larynx. Later on the swollen tissues exhibit fluctuation in one place and pus finally breaks through in one or more places.

Every form of parotitis generally takes a favorable course. During the course of parenchymatous inflammation, the swelling goes down gradually and disappears rapidly after pus has been discharged spontaneously or evacuated surgically. The wound usually closes rapidly; frequently, however, a salivary fistula remains, which closes up finally under the proper treatment. Exceptionally the facial nerve of the same side may become paralyzed in consequence of compression by the swollen gland or of an extension of the inflammation along the nerve trunk.

Epizootic parotitis is accompanied by high fever; it may be unilateral or bilateral, and never results in suppuration. In cows it is sometimes accompanied by a mild catarrhal mastitis (Bissauge).

**The chronic inflammation** presents a firm, tough swelling of the parotid which is not painful. In making a diagnosis it is necessary to exclude swellings of a different nature (true tumors, etc.) which may occur in this region.

**Diagnosis.** Acute parotitis may be confounded with an inflammation of the neighboring connective tissue or of the re-

gional lymph glands. However, the swelling in these cases is more diffuse and does not reach up to the ear. The differential diagnosis may become very difficult since parotitis sometimes spreads to the neighboring connective tissue and may also be complicated by an affection of the lymph glands. Acute tuberculosis of lymphatic glands of the parotid region occurs in cattle and frequently also in hogs, and this fact must be considered in diagnosis. Pure cases of parotitis may be distinguished from pharyngitis by the absence of cough and nasal discharge and usually also of disturbances of deglutition; further, by the superficial character of the swelling in the parotid region. In catarrh of the guttural pouch there is a nasal discharge, while the parotid remains normal in size and consistency.

**Treatment.** To counteract the inflammatory swelling, Priessnitz' applications are indicated, possibly with carbolized instead of with ordinary cold water. Ointments favoring absorption, such as iodoform-, iodinevasogen-, camphor- or mercury-salves, are beneficial. Abscesses must be opened, the sooner the better. Chronic swellings may be treated by strongunctions, injections of tincture of iodine, or of Lugol's solution; iodide of potash may be given internally. (Iodine treatment is particularly successful in actinomycosis.)

**Literature.** Bissauge, Rec., 1897, 289.—Busquet & Boudeaud, Vet. Jhb., 1903, 129.—Labat, Rev. vét., 1891, 57.—Meyer, Ö., Vj., 1855, 87.—Prietsch, S. B., 1905, 81.—Renault, Rec., 1830, 305.

**Inflammation of the submaxillary gland**, almost without exception, follows upon the penetration of foreign bodies, especially parts of food in straw feeding (Albrecht) into Wharton's duct. More rarely does it follow upon parotitis or stomatitis (authors' case).

The disease is most common among cattle, less among horses and least among dogs. One can feel the swollen, painful submaxillary gland upon the inner side of the angle of the maxilla. The margins of the meatus of Wharton's duct are often reddened and swollen; upon pressure on the duct, pus is frequently discharged. On the side and under the tongue a fluctuating or purely edematous and painful swelling may be recognized (Ranula Inflammatoria). More or less profuse salivation and disturbances of mastication likewise exist. The inflammation often leads to suppuration and more rarely to partial necrosis of the glandular tissue. Pus, usually fetid in character, breaks ordinarily into the buccal cavity, more rarely towards the outside; the disease then ends in recovery in one to two weeks.

The treatment is similar to that recommended for parotitis.

**Literature.** Albrecht, W. f. Tk., 1890, 317.



## DISEASES OF THE PHARYNX.

## 1. Pharyngitis.

(*Rachenentzündung, Halsentzündung, Schlundkopfentzündung, Halsbräune* [German]; *Angina*.)

As pharyngitis are designated all inflammations, which are partly catarrhal, partly deeper seated and penetrating even into the submucous tissue of the pharyngeal mucosa, including the soft palate and the tonsils. Such inflammations produce in man a constricting sensation of the pharynx during deglutition and they are therefore also called angina or synanche.

**Occurrence.** Pharyngitis is observed most frequently in horses and hogs, more rarely in cattle and still more rarely in sheep or carnivora. Among the first mentioned animals it occurs quite often enzootically, among the latter more rarely. Angina among chickens has been observed by Albrecht.

**Etiology.** Of external causes of acute pharyngitis, must be mentioned mechanical injuries by swallowing rough, pointed particles of food or other foreign bodies which penetrate the wall of the pharynx during deglutition or which injure the mucosa, and so form a portal of entrance for pyogenic bacteria. Hot feed or hot drinks, acrid, caustic materials may likewise produce an inflammation of the pharyngeal mucosa. This may also be produced by the inhalation of gases, hot air, or hot smoke during a conflagration. Sometimes gastrophilus larvæ produce pharyngitis either by direct mechanical irritation or by forming a portal of entrance for infecting bacteria. (Gastrophilus larvæ may also be an accidental finding in pharyngitis, as shown by the observations of Lichmann & Buffington.)

Colds sometimes play an important rôle. This is suggested by the frequency of the affection during the cold season when the animals are frequently exposed to the influence of sudden changes of temperature. Pharyngitis may appear, particularly in horses, a few days after exposure of heated animals to cold rain or sharp wind, or when cold air has come directly in contact with the pharynx. The ingestion of very cold, hoar-froste-

or frozen feed, or of very cold water may have a similar influence. These influences are particularly potent in cattle when, during winter, they are kept in warm barns and are taken out to drink very cold water.

Infection undoubtedly often plays an important rôle. Even in those cases when the disease follows immediately upon a cold, the latter very probably only forms the predisposing cause. Suggestive for an infection as the cause is the fact of the enzootic appearance of the disease in certain localities with a tendency to progress from animal to animal. The bacteria concerned may be those which occur in the buccal cavity and pharynx of otherwise healthy animals (streptococci, bacillus necrophorus, bipolar bacteria) and which may only occasionally exert a morbid effect after the resistance of the mucosa has been lessened by noxious external influences.

The bacillus necrophorus deserves first consideration as it has repeatedly caused enzootics of angina in hogs (Johné & Meyfarth, Kitt, Schleg) and also appears to have played a rôle in diphtheritic pharyngitis in cattle as observed by Mayr, Strebel, Prietsch. Similar anginas have been observed in sheep (Roche-Lubin, Diem) and in dogs or cats (Robertson, Gray, Symes).

Young dogs sometimes develop an enzootic pharyngitis during the first weeks, which may pass into pyemia (Friedberger & Fröhner). In a case of croupous pharyngitis of a dog, Ball demonstrated streptococci which he believed to be the cause of the disease.

Secondary pharyngitis, as a part of the clinical picture of specific infectious diseases, frequently appears either sporadically or enzootically. Such diseases are strangles, influenza of the horse, buffalo plague, hemorrhagic septicemia of cattle and of swine, fowl cholera, anthrax, diphtheria, purpura hemorrhagica, variola, etc. Pharyngitis due to the bacillus suissepticus (septicemic angina) occasionally appears in enzootic form (Graffunder & Schreiber, Pr. Vb.).

Inflammatory processes of neighboring tissues such as the nasal and buccal cavities, the larynx, the air sac, the esophagus, and of the bones of the face not infrequently extend to the mucosa of the pharynx.

**Chronic pharyngitis** is quite common. It is, however, of no great importance from a clinical standpoint, since it does not lead to important pathologic disturbances. It generally develops after an acute pharyngitis or after repeated, long-continued irritation.

**Susceptibility.** Horses and swine are most prone to develop disease of the pharynx. The marked difference in the predisposition of various groups of animals partially depends, perhaps, upon the fact that the tonsils of horses and swine possess several foramina coeca, those of other animals only single ones. Hence, it appears that the tonsils of horses and swine

may more easily be invaded by pyogenic and other microorganisms than those of other animals.

**Anatomical Changes.** Catarrhal inflammation (pharyngitis catarrhalis) affects the mucosa only; this appears reddened diffusely or in spots, swollen, covered with a tenacious mucus; the lymph follicles and the mucous glands are swollen and the submucosa tissue may be in a condition of edematous infiltration.

In severe cases the highly swollen mucosa may become ulcerated and the submucous tissue then presents a thickened, gelatinous mass, containing streaky extravasations of blood, or purulent infiltrations (ph. purulenta s. phlegmonosa). Submucous abscesses may be formed in certain places, particularly in the retropharyngeal region (abscessus retro- or peripharyngeales). These then protrude into the pharyngeal cavity, encroach upon it, or may even obliterate it completely. After the opening of the abscess into the pharynx or towards the outside, there may exceptionally remain a pharyngeal fistula. After injury of the pharynx or after malignant infection, the abscess frequently contains fetid, ichorous masses, sometimes mixed with remnants of food. Retropharyngeal abscesses may also be formed in consequence of suppuration of the post-pharyngeal lymphatic glands. Such abscesses are formed in the horse in strangles or influenza. In the dog, retropharyngeal abscess often appears simultaneously with purulent parotitis.

In some cases pseudomembranes form on the mucosa; they may consist of a deposit of exudate (ph. membranacea s. crouposa) or they may contain necrotic tissue (ph. diphtheritica).

The tonsils are always more or less swollen, their crypts filled with a thick purulent caseous, ill-smelling material. The soft palate is likewise swollen; the connective tissue between the bundles of muscle fibers is infiltrated edematously.

In pharyngitis in hogs due to the bacillus neerophorus the tonsils are much swollen, dirty grayish-yellow; the crypts are filled with dry caseous material, the lymph follicles in the neighborhood and in the mucosa of the pharynx in general are necrotic, the surfaces coated everywhere with masses of fibrin. The necrosis sometimes spreads to the surface of the tongue, while the submucous tissue of this organ and of the pharynx, and likewise the muscles become infiltrated edematously. The neighboring lymph glands show acute swelling.

In **chronic pharyngitis**, the mucosa is thickened, particularly on the posterior wall, and is bluish-red; the lymph follicles and mucous glands form nodules of the size of a lentil (ph. granulosa). The muscles of the pharynx become atrophic and the connective tissue proliferated (Trasbot). In horses the tonsils may increase in size to form purplish masses of the size of a finger (Kitt).

**Symptoms.** In acute pharyngitis the ingestion of food occurs slowly and carefully and there is difficulty in deglutition.



The food is masticated slowly and the head and neck are stretched out in swallowing. The animals are sometimes restless and dogs and hogs occasionally cry out. Sometimes food is expelled after repeated unsuccessful attempts at swallowing, to be again taken up later. Finally, the animals do not touch their food any more, and rather starve than be again and again exposed to the pain of swallowing. The ingestion of fluid, while easier, also causes pain, so that the patients may even refuse to drink, but only irrigate or wash out their mouth in water that is held before them. The patients sometimes make swallowing movements, probably in consequence of the inflammatory irritation and of the masses of mucus which are collecting. Vomiting is brought about similarly, particularly in dogs and swine.

In a large number of cases, however, usually only in horses, ingested water is partly expelled through the nasal cavities (regurgitation). The cause of this symptom lies in the edematous infiltration which, in the severer inflammations, develops in the submucous connective tissue of the soft palate, the lower portion of the pharynx and between the muscles of the roof of the tongue. This interferes mechanically with deglutition and with the proper contraction of the muscles.

Nasal discharge occurs during the acute course of the disease, and the mucous secretions usually mixed with particles of food appear in large amounts at the anterior nares. A low position of the head in horses sometimes increases the nasal discharge. More or less salivation is likewise present. Increased tenderness of the pharynx is manifest in a stiff, stretched position of the head and, on moving, the animals hold the head and neck in this stiff position. They try to escape palpation and even a careful touching of the region of the larynx excites manifestations of pain and often leads to a convulsive cough, accompanied by a peculiar, snorting sound. The pharyngeal region frequently appears swollen, tender and hot. The swelling is particularly noticeable in hogs and the mucosa is purplish. The submaxillary and upper salivary glands are swollen, although they may not be palpable, in consequence of edematous infiltration. Sometimes swelling of the salivary glands is noticeable. In calves, sheep, dogs, cats and fowls, direct inspection of the pharynx shows the swollen and intensely reddened mucosa, covered with pseudomembranes. These conditions are particularly noticeable on the soft palate and on the pillars of the fauces. The tonsils are likewise swollen and they may have become so much approximated to each other that there is only a slender cleft left between them. The pharyngeal wall is covered with a profuse mucoid, or muco-purulent secretion.

Cough is present in all cases, either after deglutition or in regular attacks. The animals then cough up a large amount of secretion, which is often mixed with particles of food. Laryngitis, which really is the cause of the cough, may become dangerous in consequence of edema of the epiglottis or of the



arytenoid cartilages. In such cases respiration becomes difficult. Deep inspiration after each cough, and later on every inspiratory effort, is accompanied by a snorting or whistling sound. The animal has attacks of dyspnea and the latter may become fatal. These respiratory disturbances are particularly well marked and common in the hog. Elevation of the temperature exists in the majority of cases. In pharyngitis following a cold or an infection, fever is usually present from the start and the latter may precede the other symptoms for one to two days (Fig. 32).

Pharyngitis from traumatic or chemical causes usually begins without an elevation of temperature, and fever only appears later after ulceration or pus formation has occurred. An elevation of temperature recurring during the later course of the disease points to a penetration of the process into the

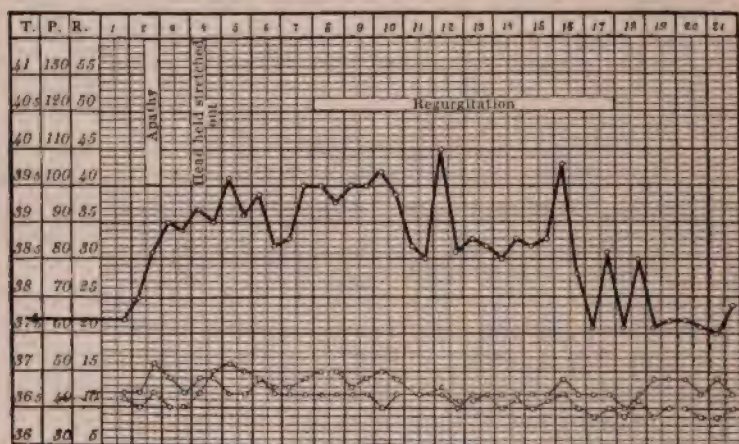


Fig. 32. Fever curve in a case of pharyngitis in a horse.

deeper tissues. In these cases the fever then generally persists until the shedding of the necrotic tissues or until the spontaneous or artificial opening of an abscess, when the temperature usually goes down within one to two days.

The appetite may be permanently retained in those cases which are not accompanied by fever at all or only by a moderate elevation of temperature; but even in these cases the nutrition usually suffers in consequence of the difficult deglutition.

The urine, in insufficient nutrition assumes an acid reaction even in herbivora, and it may be quite thin for days. The leucocytes are increased in ichorous necrosis of tissues. Meyer demonstrated an increase in the number of leucocytes of the blood in acute pharyngitis.

The symptoms of **chronic pharyngitis** are in nature similar to those described above; they are, however, milder in character. Deglutition is difficult but not impossible; the pharynx

geal region is swollen moderately or not at all; it is not very tender; the ingestion of food is frequently interrupted by attacks of cough and the anterior nares discharge a tenacious mucoid or mucopurulent secretion.

**Course.** Primary acute pharyngitis usually takes a favorable course, except in hogs, where the disease usually leads to a fatal issue. The symptoms appear in rapid succession, so that they are fully developed in two to three days; they then remain at their height for an equal period of time and recede gradually, so that at the end of the first, or during the second week, complete recovery occurs.

The course is more protracted when ulceration or suppuration of the deeper tissues occurs. In these cases all symptoms persist for a longer time or even increase in intensity. The fever goes up, remains stationary or becomes remittent until the shedding of the necrotic tissue occurs or the abscess opens into the pharynx or into the outer world. After opening of the abscess and the discharge of pus, the condition of the patient rapidly ameliorates and recovery takes place. A pharyngeal fistula, which may have formed, generally closes only after a long period of time. Occasionally a thickening in the pharyngeal wall may be formed, which may permanently disturb deglutition and respiration, particularly during work (Wetzel). A catarrh of the guttural pouch may occur in horses; in hogs, a tough, not painful, swelling may be formed after the opening of a number of small subcutaneous abscesses, which permanently interferes with deglutition and respiration. Paralysis of the pharynx may also occur, but may again disappear after a prolonged period.

A fatal issue is generally due to complications. Edema of the larynx, in the absence of proper aid, may lead to suffocation. An occurrence, which is not infrequent in horses, is aspiration pneumonia due to swallowing or inspiration of pus of an abscess breaking into the pharynx. Inflammation may also spread into the loose connective tissue in the mediastinum, and then a pleuritis or pericarditis closes the chapter (Cadéac). In phlegmonous pharyngitis septic material may be absorbed from ulcerations and lead to general sepsis or a fatal hemorrhage may occur at the base of an open ulcer (Wakefield, De Jong, Monod).

**Diagnosis.** The clinical picture of pharyngitis is, as a rule, quite characteristic, so that diagnosis offers no difficulties. Foreign bodies which have become wedged in the pharynx, generally in carnivora and cattle (Fiebiger has seen two such cases in horses), can best be detected by inspection and internal manual palpation. Tumors of the pharynx are usually not painful, the signs of an acute affection are missing and internal palpation reveals the tumor. In paralysis of the pharynx there is no



tenderness and the peculiar position of the head is absent. Diseases of the central nervous system will lead to other paralysis besides that of the pharynx; and in cases of meningitis there are mental disturbances. In obstruction of the esophagus no changes are found in the pharynx and regurgitation during the act of deglutition occurs later; a sound will find the obstruction in the esophagus. Parotitis does, generally, not lead to regurgitation, and is not accompanied by nasal discharge. If pressure is made on the lower wall of the pharynx (base of the tongue), pain is not elicited. Catarrh of the guttural pouch generally causes a unilateral swelling in the upper portion of the parotid region; pressure on the latter usually causes nasal discharge; pressure on the lower pharyngeal wall does not elicit pain. Since pharyngitis may be a part of the picture of a general infectious disease, one must, in the beginning, think of this possibility. In horses one must think of strangles; in cattle and swine, of hemorrhagic septicemia, hog cholera, or anthrax.

The determination of the nature of pharyngitis is usually very difficult, except in smaller animals where inspection permits a good survey of the anatomical changes. It may be said, in general, that high fever persisting during the course of the disease, intense and increasing swellings of the pharynx, speak for a deeply penetrating inflammation. Gangrene of the mucosa, phlegmonous or diphtheritic inflammations, aside from their local manifestations, are characterized by a fetid smell of the saliva and the expired air. Coughed up masses of fibrin point to croupous pharyngitis. In proper cases one may use in horses the rhinolaryngoscope of Polansky & Schindelka, which occasionally makes it possible to recognize swellings of the mucosa, hemorrhages, small abscesses, etc. In difficult respiration this instrument cannot be used.

**Prognosis.** The prognosis of primary uncomplicated pharyngitis is favorable; however, it becomes the more unfavorable the more the symptoms point to phlegmonous, diphtheritic or purulent inflammation. Even affections which are mild at the start, may in their further course assume a malignant character, particularly in horses, where aspiration may easily occur; hence a guarded prognosis is advisable during the early stages.

**Treatment.** Considering the increasing tenderness of the mucosa and the difficulties in deglutition, the food must be soft, mushy or fluid in consistency. In herbivora, the best nourishment is green feed, well wetted hay, flour or bran mash thin in consistency, while hogs and carnivora are best fed with milk. Horses suffering from intense difficulty in deglutition should at first not be fed even with entirely fluid feed and water; thirst can be alleviated by repeated injections of lukewarm water into the rectum. The patient should fast from one to two days and



tions may sometimes give relief in combination with Priessnitz' applications (oil of mustard in 6 to 8% alcoholic solution, red biniodide of mercury ointment 1:4).

Abscesses should, if possible, be opened early; it is sometimes possible in cattle to open an abscess, previously located in the pharynx by palpation, with a knife introduced into the mouth (Macgillivray, Cunningham). A pharyngeal fistula which has eventually formed may be made to heal within a few weeks by repeated disinfection. Tracheotomy must not be delayed in the presence of intense dyspnea or edema of the larynx. The use of internal medicines usually appears superfluous and may even be dangerous. Should it however become necessary, such medicines should be used by rectal or by subcutaneous injections.

Chronic pharyngitis in small animals may be treated with local applications of tincture of iodine (Tinct. Iodi., Tinct. Gallarum  $\bar{a}\bar{a}$ ), chloride of iron (1:6), iodine or tannic-acid-glycerine, insufflations of boracic acid or tannic acid with sugar ( $\bar{a}\bar{a}$ ). In large animals treatment must be limited to irritant inunctions and Priessnitz' application.

**Literature.** Albrecht, M. t. W., 1909, 26.—Ball, J. vét., 1906, 449.—Buffington, Am. v. R., 1905, 37.—Cunningham, Vet. Jhb., 1906, 161.—Diem, W. f. Tk., 1897, 339.—Fiebiger, Z. f. Tm., 1902, VI, 443.—Graffunder & Schreiber, D. t. W., 1902, 471.—de Jong, D. Z. f. Tm., 1892, XVIII, 306.—Kitt, Münch., Jhb., 1893-94, 81.—Lichmann, O. M., 1893, 169.—Lövy, Vet., 1892, 443.—Meyer, Z. f. Tm., 1906.—X, 1.—Nikolski, Vet. Jhb., 1886, 81.—Preis, Z. f. Tm., 1898, II, 62.—Pr. Vb., 1900, II, 9.—Sequens, Vet., 1894, 504.—Sonin, Vet. Jhb., 1888, 81.—Wetzel, A. L., 1907, 18. (See also literature on epizootic laryngo-pharyngeal catarrh.)

## 2. Paralysis pharyngis.

(*Paralysis of the Pharynx, Schlundkopflähmung* [German].)

**Etiology.** Paralysis of the pharynx is especially seen as a part of the clinical picture of bulbar paralysis in disease of the central nervous system (meningitis, meningitis cerebrospinalis enzootica, progressive bulbar paralysis, tumors), in the course of certain infectious diseases (rabies, acute infectious bulbar paralysis) and intoxications (botulism) and also, as is claimed, after poisoning with some fungi.

Occasionally it develops secondarily after acute pharyngitis (Bongartz) probably due to secondary affection of the glosso-pharyngeal nerves or of the branches of the pneumogastric nerve which supply the muscles of the pharynx. (Zschokke saw a case of paralysis of the pharynx caused by a perineuritis after infection of the guttural pouch of a horse with hyphomycetes.) Tumors compressing the pneumogastric nerve may exceptionally produce paralysis of the pharynx. Sometimes the latter may appear as a primary condition.

**Symptoms.** Paralysis of the pharynx makes deglutition impossible, consequently food and water taken up are again

voided by the mouth, or expelled through the nose. The morsels of food cannot descend into the esophagus, though attempts at deglutition occur accompanied by loud sounds. Salivation is always profuse, because the saliva cannot be swallowed and hence collects in the buccal cavity. Portions of food or whole mouthfuls may become wedged in the pharynx; parts may get into the larynx and trachea and cause gangrene of the lung. Introducing the hand into the pharynx of larger animals does not produce any contraction of the pharyngeal muscles (Zürn). Whistling and snorting noises accompany the movements of the animals and may even be heard during rest; the disturbances of regurgitation diminish the capacity for work.

Straub and Friedberger have seen horses unable to swallow water but able to swallow dry food in a perfectly normal manner. In these cases, the causes of which could not be ascertained, the remedy consisted in mixing the dry feed with water so that the system received a sufficient amount of fluid.

**Diagnosis.** A careful examination must exclude all other diseases of the pharynx (wedged foreign bodies, tumors, retropharyngeal abscess), occlusion or compression.

**Treatment.** Paralysis due to local inflammation sometimes subsides without any treatment at all (Dieckerhoff). The animals must, however, in all cases be fed artificially (see page 123). Irritant inunctions and nerve tonics by subcutaneous injection are frequently used. Recovery in horses was seen by Bongert after daily injections of 0.05-0.06 gm. strychnia; Langer, 0.03 strychnia; Sonnenberg after one injection of 0.025 gm. arecoline. Sometimes, however, it is necessary to slaughter the animals in order to recover part of their value.

**Literature.** Besnoit, *Rev. vét.*, 1903, 10.—Bongartz, A. f. Tk., 1881, VII, 485.—Dexler, *Nervenkrkh. d. Pferdes*, 1899, 30.—Langer, *Z. f. Vk.*, 1890, 417.—Sonnenberg, B. t. W., 1906, 858.—Straub, *Rep.*, 1858, 26.—Zschokke, *Schw.*, A., 1907, XLIX, 313.—Zürn, *D. t. W.*, 1905, 62.

**Enzootic Paralysis of the Pharynx in Cattle.** This name was given by Dieckerhoff to a disease of cattle originally called meningitis boum enzootica by several authors (Vogel, Utz, Mayer, Zipperlen and others). Dieckerhoff believed that the disease is caused by an infectious substance contained in the food, because this assumption would explain its enzootic nature. It is not spread to neighboring farms. The disease has been noticed particularly in Germany, although Andersen and Berg have seen it frequently in Denmark. Its nature and its relation to enzootic cerebrospinal meningitis are not definitely known.

To this group may also belong an enzootic paralysis of the pharynx frequently seen in cattle, less frequently in sheep and horses, and believed to be due to the ingestion of food much contaminated with rust fungi (Johne, Herele, Adam, Brandt and others).



irrigation with solutions containing common salt, vinegar or ammonia or in the inhalation of turpentine vapors.

Tokishige found filiform worms in the pharynx of a horse dead from pharyngitis, which he thought to be *Dispharagus reticulatus*.

#### 4. Tumors of the Pharynx. Tumores intra- et peripharyngeales.

**Occurrence.** Tumors of the pharynx and its neighborhood are rare in domestic animals. Least frequently seen are such true neoplasms as papilloma, sarcoma, carcinoma, lipoma, also retention cysts; connective tissue hypertrophy of the mucosa of the pharynx is seen after an acute pharyngitis (Wetzel). Actinomycomata or tuberculous growths are seen more commonly in cattle.

According to Zimmer's statistics seventy-three cases of tumors of the pharynx in cattle were divided as follows: Fifty-four cases of actinomycosis, five cases of tuberculous growths, seven cases of dermoid cysts, four cases of fibroma, two cases of colloid cysts, one case of melanoma. Out of 4,708 cases of profound tuberculosis Rasmussen found retropharyngeal lymph glands in a condition of tuberculous degeneration in 3,245 cases (68.93%); the gland below the parotid in 228 cases (4.84%); and the tonsils only in fifty-two cases (1.1%).

As has been shown by the fundamental work of Mörkeberg, actinomycosis usually develops in the upper, posterior parts, more rarely in the lateral walls of the pharynx, that is either in its mucosa or in the neighboring tissues. These actinomycotic masses then show a tendency to project polyplike into the pharynx. Tuberculous tumors, on the contrary, arise outside of the pharynx and only make its wall protrude in a tumorlike manner. The root of the tongue is usually the seat of retention cysts, also the anterior surface of the epiglottis or a place between the latter and the root of the tongue. True tumors usually arise in the upper posterior portion of the pharynx.

Intrapharyngeal tumors usually become pediculated in consequence of traction made in deglutition.

**Symptoms.** The sessile tumors produce increasing difficulty in swallowing, but without pain; later dyspnea and rattling, whistling and snorting noises are heard, when the tumor encroaches upon the posterior nares and the larynx. Difficulty in deglutition and respiration is increased when the head is bent forward or sideways. Stenosis of the posterior nares causes labial breathing in all animals (bulging of the cheeks during expiration) which disappears when the mouth is opened.

Smaller, pediculated tumors cause difficulty in deglutition or respiration as well as rattling sounds only occasionally, namely, when they become displaced into the pharynx, larynx or towards the choanæ, in deglutition, strong inspiration and in certain positions of the head, until they are again expelled by a strong expiration or a movement of the head. It may, however, occur occasionally that such an attack brings about suffocation of the animal. As the tumor grows the attacks

become more frequent and lead finally to permanent difficulty in deglutition and respiration.

Discharges from the nose are only observed in part of the cases, such discharges are sometimes fetid and mixed with fragments of tissues. Epistaxis occasionally occurs; dogs sometimes vomit and the tumor may then be visible in the buccal cavity.

With the exception of those on the posterior surface of the soft palate, tumors of the pharynx in smaller animals can be seen on inspection; in horses, however, only with the aid of the rhinolaryngoscope. With the hand introduced into the pharynx even those tumors can be palpated which arise in the neighborhood of the posterior nares.

Peripharyngeal tumors when present produce visible changes in the pharynx.

Swelling of the submaxillary, also occasionally of the peripharyngeal lymphatic glands, occurs in malignant tumors and tuberculosis of the pharynx.

**Diagnosis.** The described disturbances of deglutition and respiration, if developing slowly, strongly suggest the presence of pharyngeal tumors; a definite diagnosis can however be made only after local inspection. When the examination of the pharynx is negative, tumors of the larynx may be discovered by laryngoscopy and it may perhaps be necessary to make an exploratory laryngotomy.—Tumors of the larynx may also be distinguished by the fact that compression of the larynx increases the dyspnea; this is not the case in tumors of the pharynx. However, exploratory tracheotomy may be necessary to settle the point.—Nasal stenosis can easily be excluded by a local inspection.—Retro- and peripharyngeal abscesses furnish a similar clinical picture during a protracted course, but local examination and the history of the case permit a correct differential diagnosis. In making the latter the following other conditions have to be considered: Chronic disease of the pharynx, and of the guttural pouch, spasm of the glottis, stenosis of the bronchi, and in the case of dogs cardiac dyspnea in uncompensated valvular disease of the heart, which likewise leads to attacks of rattling respiration.

The determination of the nature of the tumor requires a careful examination of the pharynx and its neighborhood.

**Prognosis.** This depends upon the seat and the nature of the tumor. If the latter can be removed, the animal can be saved; if its removal is impossible the animals die from suffocation, pneumonia or marasmus. Even tuberculous tumors removed and complete recovery may follow the operation has been purely local. Tuberculous and

**Treatment.** Pediculated tumors and those not having a broad base may be crushed or cut off after ligation of the pedicle with instruments introduced through the mouth. A preliminary tracheotomy is however usually necessary. Iodide of potash often proves of no value in the treatment of actinomycotic tumors of the pharynx. (Details about the operative treatment of pharyngeal tumors may be found in the publications of Mörkeberg.)

**Literature.** Mörkeberg, Z. f. Tm., 1907, XI, 63 (Lit.).—Rasmussen, B. t. W., 1906, 848.—Wetzel, A. L., 1907, 3.—Zimmer, B. t. W., 1891, 376.



## SECTION IV.

### DISEASES OF THE ESOPHAGUS.

#### 1. Inflammation of the Esophagus. Oesophagitis.

(*Entzündung des Schlundes* [German].)

**Etiology.** Irritating, caustic substances or rough, pointed bodies, which are ingested with food or water, or improperly selected medicines, may get into the gullet and cause catarrh or a rather penetrating inflammation of the esophagus. Their injurious influence here becomes more rarely manifest than in the buccal cavity or pharynx, because the esophageal mucosa is less sensitive and better protected by a thicker epithelial covering, and also because irritating matters very rapidly pass through the gullet. These morbid conditions are met with after the ingestion of very hot distillers' slop, after the administration of ammonia, tartar emetic, after poisoning with acids or alkalies or after greedy swallowing of rough fodder, fragments of bones mixed with meat, foreign bodies which become wedged in the gullet, or after injury by the esophageal sound. Finally, there are traumatic influences acting from without which may become the cause of the inflammatory process.

The disease may be secondary to inflammatory processes of the pharynx or the stomach, especially if these are due to a general infection (foot-and-mouth disease, variola, diphtheria, rinderpest, etc.).

**Anatomical Changes.** In superficial inflammations the epithelial covering is missing either in irregular patches or sometimes to a larger extent, or even along the whole length of the esophagus (Berthéol). The mucosa appears dark red on the uncovered portions, occasionally hemorrhages are seen here; in the other portions the epithelial layers are loosened and can be removed easily; the submucous connective tissue exhibits a more or less intense edematous infiltration. In severe cases the wall of the esophagus is thickened, swollen and the subcutaneous and intramuscular tissue is the seat of a gelatinous or even purulent infiltration. Cattle have developed croupous inflammations after the instillation of spirits of ammonia (Lemaire).

Chronic catarrh leads to a marked thickening of the epithelial layers, occasionally also to papilliferous proliferations.

**Symptoms.** A very superficial catarrh of the esophagus usually escapes notice. In somewhat more intense cases the food is swallowed with a great deal of effort. The animals move the head restlessly from side to side in swallowing, stretching and bending the neck, horses paw with their front feet and their faces express anxiety. Sometimes one may observe how a morsel becomes wedged in the esophagus, occasionally directly below the pharynx. Such morsels then become dislodged toward the pharynx by antiperistalsis and are expelled through the mouth or nose. Deeply penetrating inflammations lead to a mucous secretion or to a bloody discharge from the mouth or nose independently of the ingestion of food. In such cases food is refused. Tubular croupous masses are sometimes expelled in croupous inflammation. Increased tenderness of the esophagus is shown by the animals when pressure is made over the left jugular depression. One may sometimes see here spontaneous undulating motions followed by the expulsion of mucus or food from the mouth. In more intense inflammation of the esophagus, particularly in carnivora, vomiting occurs. Difficulty in deglutition greatly interferes with the nutrition of the animals and the inflammatory process may cause an elevation of temperature.

**Course.** Simple catarrh ends in recovery after one to two weeks, while deeply penetrating inflammations lead to cicatricial contractions of the esophagus or to a purulent infiltration in the surrounding connective tissue. The infiltration spreads along the large vessels towards the thoracic cavity and leads to the formation of abscesses. Exceptionally a perforation of the esophagus occurs. All these complications are indicated by an inflammatory swelling in the region of the left jugular vein. If perforation occurs in the thoracic portion of the esophagus pleurisy follows.

**Treatment.** One should attempt to ameliorate the inflammatory process by the administration of small pieces of ice, by cold water, cold milk, mucilaginous or astringent fluids ( $\frac{1}{2}$  to 1% tannic acid in linseed infusion). Cold applications on the region of the esophagus should also be made. If the inflammation is due to corrosive poisons, antidotes like weak acids or alkalis in mucilaginous milk should be administered. When the pain is intense narcotics are indicated (morphine subcutaneously, chloral hydrate, tincture of opium in the drinking water). If the animals still persist in refusing to take food, artificial feeding per rectum becomes necessary.

**Literature.** Eichenberger, D. Z. f. Tm., 1885, XI, 111.—Guilmot, Ann., 1854, 341.—Dohne, S. B., 1879, 46.—Koch, B. t. W., 1889, 229.—Lebel, Rec., 1864, 355.—Lemaire, Ann., 1860, 544.—Renault, Rec., 1834, 561.

## 2. Spasm of the Esophagus. Oesophagismus.

(*Krampf des Schlundes* [German].)

Spasm of the esophagus consists in a morbid contraction of its muscles, which occurs in paroxysms, without organic disease or obstruction. These latter conditions also lead to spasm of the muscularis of the esophagus. Esophagism, as a disease per se, is rare among domestic animals.

**Etiology.** Primary disease of this kind is very rare, and it then occurs upon a neuropathologic basis. It has been observed without any special cause only a few times in nervous horses of quick temper. Such animals may have an attack upon drinking cold water or upon pressure upon the esophagus. Nothing definite is known whether in these cases disease of the pneumogastric nerve or transitory malposition of the esophagus may play a rôle.

Horses sometimes exhibit esophagism after the injection of morphine or after chloral hydrate or chloroform narcosis (Fröhner, Venneholm). In a case of this kind Plósz could demonstrate the obliteration of the esophagus by a morsel of food. A similar morbid condition occurs very rarely in adult cattle (Ries) and somewhat more frequently in calves (Trousier). Esophagism occurs secondarily in the course of epilepsy, tetanus and rabies.

**Symptoms.** When the spasm begins, the animal suddenly becomes very restless and betrays great anxiety; if it happens to be feeding when the spasm comes on, the ingestion of food ceases, yet empty mastication continues, and a foamy saliva collects in the mouth. The animal stretches its feet out and tries to swallow the saliva while the head is strongly stretched or bent. At the same time, the undulatory motions of the esophagus become visible in the left jugular region. However, swallowing of the saliva can take place only incompletely, and it is expelled through the mouth and nose by antiperistaltic movements. If the spasm occurs during the ingestion of food, the saliva is at first mixed with food particles, later on it becomes pure and does not give an acid reaction like expelled gastric contents. Along the left jugular depression the cordlike gullet can be felt distinctly, pressure upon it excites pain. Sometimes a moderate enlargement, filled with air, saliva and mucus, is formed above the spasmodically closed section (Friedberger).

The attack may last from a few minutes to several hours; it may or may not recur. In a case reported by Cadéac, the sick horse had several such attacks every week for a year and a half, and these could be produced at will by feeding short cut dry feed or by the administration of cold water. Roy, on the



other hand, saw five attacks in five years; they always lasted five hours and were followed by respiratory difficulties lasting for twenty-four hours. The disease always ends in recovery.

**Diagnosis.** On account of the disturbances of deglutition and the subsequent vomiting, esophagism may be confounded with inflammation, stenosis or obstruction of the esophagus. However, the sudden appearance, the likewise rapid disappearance of the attack, the perfectly normal condition of the animal between the attacks furnish enough data to distinguish this affection from stenosis or inflammation. Primary esophagism may be distinguished from mild cases of obstruction of the gullet in the thoracic portion, which disappear spontaneously after a short time, by the use of the sound (perhaps after the preliminary application of narcotics). If no impediment is found and if the attacks still persist, the diagnosis is, of course, primary esophagism. It is also possible that during very greedy feeding, a morsel may become lodged in the caudal portion of the esophagus, this subsequently may pass on, yet the described symptoms may persist a while, and a veterinarian not knowing the history of the case may think of primary spasm of the esophagus (Cagny, Johne). Indeed, esophagism is, as a rule, due to a temporary obstruction or to a superficial injury.

**Treatment.** The disturbed condition of the nerves calls for the use of narcotics, such as morphine subcutaneously (0.04-0.6 gm. for horses; 0.01-0.1 gm. for smaller animals); chloral hydrate per rectum (25-50 gm. for large animals; 0.05-0.5 gm. for smaller animals); during the intervals between the attacks bromide of potash internally (for horses, in daily doses of 20 gm.). This treatment is usually successful.

**Literature.** Bournay, Rev. vét., 1898, 204.—Cadéac, J. vét., 1888, 618.—Friedberger, Münch., Jhb., 1890-91, 60.—Fröhner, Monh., 1897, VIII, 484, 1898, IX, 345.—Johne, S. B., 1879, 45.—Ries, Rec., 1897, 228.—Roy, Rev. vét., 1898, 286.—Winke, Holl. Z., 1905, XXXII, 56.

### 3. Paralysis of the Esophagus. Paralysis oesophagi.

(*Laehmung des Schlundes* [German].)

**Etiology.** Paralysis of the esophagus occurs usually together with paralysis of the pharynx, and is due to the same causes as the latter (see page 214). There have, however, been described a few cases of primary paralysis of the esophagus in horses, the nature of which has not been cleared up. Moeller saw this affection a few times after resection of the arytenoid cartilages, sometimes as a temporary, sometimes as a permanent condition. He found marked proliferation of connective tissue around the larynx in one case and thinks it may be possible that

in other cases likewise inflammatory swelling in the first portion of the esophagus might have been the cause of the disturbed deglutition. In a case of Brissot, a fall upon the left side, and in a case of Graf, a kick on the neck were the causes of paralysis of the esophagus. (In these two cases a traumatic lesion of the esophageal wall itself cannot be entirely excluded, also in the case of a horse of the Prussian army.) In this case a gelatinous bloody infiltration was found behind a portion of the gullet filled with particles of food. Puschmann and Schneider were unable to discover a cause in their cases.

Occasionally histologic changes may be discovered in the pneumogastric or recurrent nerves.

**Symptoms.** In primary paralysis of the esophagus the food is always masticated without trouble; but masses of fodder accumulate in the gullet. In this manner the esophagus forms a thick, firm or more soft, non-painful, cylindrical prominent mass in the left jugular depression. Deglutition becomes impossible and regurgitation occurs. These disturbances appear suddenly.

Disturbances in deglutition coming on after arytenectomy may disappear after a few weeks. In the above mentioned case of Brissot recovery occurred in two days, but the affection may lead to death in consequence of impeded nutrition or of foreign body aspiration pneumonia.

**Diagnosis.** A diagnosis of paralysis of the esophagus can be made after the exclusion of paralysis of the pharynx, obstruction of the esophagus, stenosis or dilation of the esophagus, contusion of the esophagus.

**Treatment.** Attempts must be made to push the accumulated, wedged-in masses of food down with the esophageal sound. The animals should then receive juicy or liquid feed only. The employment of nerve tonics (strychnine, veratrine) or of electricity, is not promising.

**Literature.** Graf, Z. f. W., 1892, 211.—Möller, Chirurgie, 1891, 190.—Schneider, W. f. Tk., 1905, 39.

#### 4. Obstruction of the Esophagus. *Obstructio oesophagi.*

(*Foreign Bodies in the Esophagus; Occlusio Oesophagi.*)

By obstruction of the esophagus is meant a sudden closure of its lumen by morsels of food or foreign bodies.

**Etiology.** Obstruction of the esophagus is seen most commonly in cattle and is caused by large, solid constituents of the food (potatoes, beets, apples) or exceptionally by entirely for-



eign body itself or by the swallowed material subsequently causes antiperistaltic movements.

Pressure of the foreign body upon the neighboring organs disturbs their function, more or less, while the compressed or directly injured mucosa of the esophagus may get into an inflammatory condition which may lead to necrosis. Inflammatory changes frequently are also produced in front of the obstruction in consequence of accumulating, decomposing materials. This diminishes the resistance of the esophageal wall and makes an acute dilation of the esophagus possible.

**Symptoms.** Pain and the peculiar sensation produced by the lodgment of the foreign body cause the animal to make strong efforts at deglutition, and to become quite restless. The animal suddenly ceases to feed, becomes restless, lowers and stretches its head and makes strong attempts at deglutition, now and then it opens its mouth, from which saliva flows abundantly; the expression of the face betrays great anxiety, the tongue protrudes from the mouth, and a convulsive cough is heard from time to time. The symptoms eventually become less marked, but are liable to recur now and then. The ingestion of food and drink is entirely abolished or the patients attempt to swallow food and water after becoming more quieted and on feeling hungry. But these are soon thrown out of the mouth and nose, although in incomplete obstruction water may get into the stomach. These attempts give us some information about the site of the obstruction. If it is located in the anterior portion, morsels of food and water at once return after an attempt at deglutition; if the obstruction is at the caudal end we may see how the swallowed material passes along the esophagus as indicated in the left jugular depression. The expulsion occurs the later the nearer to the caudal end the obstruction is located. The patients sometimes keep on swallowing in spite of the obstruction, until the esophagus is filled completely, even up into the pharynx; then the ingestion of food usually ceases, but the animals continue to masticate on an empty mouth and to make convulsive attempts at deglutition. In such cases the gullet can be felt as a firm or more or less soft cylindrical mass on the left side of the neck. Kneading of the mass may eventually excite gagging. From time to time masses of masticated food mixed with saliva are expelled from the mouth and nose; during these attacks the animals are quite restless, and the mass expelled is undigested and does not contain any free HCl. After obstruction has lasted some time, dilation of the esophagus sometimes occurs (see page 235).

The cause and site of the obstruction may be ascertained in a number of cases. Pieces lodged in the anterior portion of the esophagus may be seen in the well illuminated pharynx of the dog and cat or they may be palpated with the finger; in cattle they may be felt after the mouth has been opened, and the whole hand introduced.



If a foreign body has lodged in the cervical portion of the esophagus, the jugular depression is only bulging at a circumscribed space, either at the left or at both sides; in the latter case the bulging is more marked on the left than on the other side. By palpation one may occasionally be able to determine the consistency and shape of the body; this manipulation usually causes gagging or the expulsion of a foamy fluid.

The location of an obstruction in the thoracic portion can only be ascertained by the use of the esophageal sound or by a careful analysis of the disturbances of deglutition. Foreign bodies of a high specific gravity (especially metals), may be made visible in all animals by the aid of the Roentgen (X) rays.

In cattle and in ruminants in general, meteorism is produced the more rapidly, the more complete the obstruction and the more fermentable the previously ingested food has been. Gases can, however, be expelled partially in incomplete obstruction of the esophagus, hence meteorism is only moderate. Under these conditions the animal may begin to feed and then produce a complete obstruction by the swallowed morsels which fail to pass down.

If the obstruction lasts for any length of time in hogs, they may become moderately bloated; in such cases they stand quietly, with their head bent down, their mouth open; they are salivated, gagging occurs as if they wanted to vomit. They do not lie down, or if they do at all, for a short time only. Thirst is increased, the animals try repeatedly to drink, but the water returns to, and runs out of the mouth. If the foreign body is near the pharynx, the animals produce a yelping sound instead of a grunt.

**Course and Prognosis.** Foreign bodies lodged in the esophagus are frequently and often expelled within a short time in consequence of repeated gagging, or they are transported into the stomach or rumen by continuous strong contractions of the muscles of the esophagus. In this manner the animals recover spontaneously. This happens the more easily, the smaller, smoother and softer the foreign body is and the nearer it has become wedged to either the anterior or posterior extremity of the esophagus. However, if the efforts of the patients are futile and if they are left to themselves, there occurs a progressive deterioration of their condition. Cattle may die from suffocation within a few hours after the obstruction occurred, in consequence of the rapidly developing bloating, followed by disturbances of respiration and circulation. A rapidly fatal issue may also take place in some cases in consequence of compression of the trachea or of the nerves running along the esophagus, or of obstruction of the larynx by particles of food (Sequens).

In other cases, the animals may suffer from an obstruction for some time, even for several days, but in such cases pro-



gressive and rapid emaciation becomes noticeable and the wall of the esophagus which is compressed by the foreign body becomes necrotic. If the necrotic portion of the wall breaks into the surrounding tissues, the clinical picture terminates by a purulent or ichorous inflammation of the cervical connective tissue or, if the thoracic portion of the esophagus is involved, by a pleuritis. Inflammation and gangrene of the esophageal wall develops rapidly if the wall has been injured and a channel for infection has been opened up. Injuries produced by the introduction of the esophageal sound and in attempts at removing the foreign body may have the same effect.

Obstruction caused by morsels of food may, without veterinary interference, be recovered from speedily, i. e., if the morsel becomes softened and if it can then be removed. If, on the other hand, the morsel dries and becomes harder, it may produce dilatation or perforation with grave consequences. A fatal issue may, however, occur occasionally after removal of the foreign body into the stomach, if artificial feeding has been neglected (Drouin) or if an intense inflammation of the esophagus has occurred and now of itself causes intense difficulty in deglutition (Johne). Difficulties in deglutition and vomiting may, especially in the horse, lead to pulmonary gangrene from the start. In greedy horses, obstruction of the esophagus by too large morsels may recur repeatedly within a short time.

(Small, pointed bodies frequently perforate the wall of the esophagus and become encapsulated in the neighboring tissues without producing any marked disturbances [Bruckmüller, Kitt].)

**Diagnosis.** In obstruction of the esophagus by lodgment of a foreign body we can usually get a proper clinical history and we have the sudden appearance of the grave disturbances of deglutition. If the obstruction has occurred in the cervical portion of the esophagus, a reliable diagnosis can be made by palpation from the outside or from the pharynx. In other cases, particularly if the obstruction is in the thoracic portion, the diagnosis may meet with considerable difficulties. Spasm of the esophagus can be distinguished from the milder rapidly recovering cases of obstruction by the use of the sound, which must sometimes be preceded by the administration of narcotics (see page 223).

Stenosis or dilation of the esophagus may be excluded from the clinical history and from the observation, that soft feed and, still more, fluids will pass the gullet without difficulty some time after feeding. In paralysis of the esophagus, forced attempts at deglutition and gagging are absent.

In ruminants, acute meteorism, due to some other cause, might erroneously be referred to obstruction of the esophagus; but if not due to the latter condition, there is no disturbance and no regurgitation, and the sound meets no impediment in

the esophagus, except in ruminants; masses vomited from the stomach, smell sour, contain free HCl, and true vomiting occurs within a certain interval after feeding and with participation of the abdominal muscles. In dogs the possibility of rabies must be considered, since disturbances of deglutition are so common in this disease.

**Treatment.** Removal of a foreign body wedged in the first portion of the esophagus is best accomplished by the hand or by a suitable instrument introduced into the buccal cavity.

For this purpose the head of cattle is stretched forward, the mouth is opened by the aid of the mouth gag or simply by drawing the tongue out and to the side, next the right hand is introduced into the pharynx and the foreign body is withdrawn by the fingers of the operator. Since attempts at deglutition are frequently caused by this manipulation, and since the former might move the foreign body towards the stomach, it is best to have an assistant fix the latter from the outside. The removal of the foreign body may also be brought about in such a manner that the fingers introduced into the esophagus are spread out, the assistant pushes the obstruction towards the buccal cavity and lowers the head of the animal at the same time. In this manner the foreign body usually slides between the fingers into the palm of the hand of the operator (Rolfes). The procedure is usually applied to the standing animal; it becomes necessary only exceptionally to have the animal lie down; in such a case it should lie on the right side; horses must be thrown. Foreign bodies in the first portion of the esophagus of smaller animals are best removed by appropriate forceps.

Since the animals get excited during this manipulation and since the hand introduced into the pharynx interferes with respiration, it is necessary to act quickly. If the procedure has not been successful, it is advisable not to prolong the attempt but to wait for some time and then try again. Such repeated attempts are however not without danger and Deneubourg has seen an inflammatory cervical edema follow them, which disappeared only after one month.

When the foreign body is located in the cervical portion of the esophagus, one should always attempt to dislocate it towards the buccal cavity unless there is danger of suffocation.

For this purpose the operator grasps the neck of the animal at the sides from above with both arms, and approaches both hands immediately behind the foreign body. He tries to move it forward by alternate pressure, made best with the thumbs. If the procedure appears to be successful, it is continued until the foreign body has been pushed in front of the larynx. It must then be fixed in this position by an assistant, to be removed through the pharynx as described above. This is sometimes not necessary, because the animal begins to gag and expels the foreign body through the mouth without any further aid. To assist in the removal of the foreign body, it is advisable to press it and the larynx forward and downward and to lower the head and approach it to the thorax before the hand is introduced into the mouth. This causes a depression of the root of the tongue, and an enlargement of the space between it and the soft palate so that the foreign body can fall out more easily (Martin). It is also well to open the mouth as much as possible (Favreau). Chapellier uses two devices similar to hoof-forceps; with one he compresses the jugular gutters behind the foreign body to prevent its sliding backward; with the other he presses upon the posterior end of the foreign body and pushes it upward. The two forceps are alternately moved forward until the foreign body has been brought into the pharynx. Imminger removes foreign bodies in the most anterior portions of the esophagus by introducing a small trochar into their center from the left side of the neck; with this he then moves them up gradually.

Opinions differ as to how the head of the animal should be held during the operation. When the head is stretched and held high the esophagus forms an almost completely straight line with the pharyngeal and buccal cavity; but this also stretches and narrows the esophagus. In view of this fact it appears more advantageous to bend the head downward and backward; this certainly is



always indicated if the foreign body has already been pushed to the pharynx and is to be pressed into the pharynx or mouth. If the hand is to be introduced into the mouth, the head must be elevated. Moeller maintains that the head of the animal should not be held at all.

Some cases can be treated successfully by the subcutaneous injection of certain medicines. Hogs and dogs may receive an emetic subcutaneously (hogs, 0.02-0.03 gm. veratrine or 0.05 gm. hydrochlorate of apomorphine [Moulis], dogs, 0.01-0.02 gm. apomorphine). These emetics sometimes cause the expulsion of the foreign body after a few minutes. In other animals likewise, the following subcutaneous injections may accomplish the object, even after unsuccessful attempts at extraction, by causing energetic muscular contractions and powerful attempts at deglutition which move the foreign body into the stomach, viz: Strychnine (Maury), eserine (Clere), veratrine (Michalski), arecoline (Fröhner).

In cases where he did not succeed in removing the foreign body, Schaak proceeded as follows: He poured one quart of mucilaginous fluid into the animal and then made it run uphill; in this manner horses and cattle often swallowed the foreign body.

If the methods described are unsuccessful, the further procedure depends upon the nature of the wedged-in body. Fruit, bulbs, morsels of food, etc., in the course of time become softened in the esophagus and are finally swallowed. Since obstruction of the esophagus in cattle soon leads to bloating, one may leave the foreign body undisturbed only if meteorism has been relieved in the meantime. It is therefore necessary to perforate the rumen and it is best to leave the shield of the trochar in the wound, closing its outer opening with a cork, and to open it from time to time in order to let out the accumulating gases. It is also necessary, in the further course of the disease, to attend to the artificial feeding of the patient per rectum.

If the wedged-in body is too hard, or if its nature is unknown, if one wants to accomplish removal when other means have failed, displacement towards the stomach may be attempted by the aid of an esophageal sound, a catheter, and in an emergency, with a carefully wrapped elastic stick (stick of a whip, piece of rattan), or a piece of moistened and oiled rope with a knot at one end. The foreign body must, however, be pushed without much force and very gradually. The extraction of foreign bodies with special armed extraction forceps or the crushing of the foreign body is not to be recommended.

The propulsion of the foreign body towards the stomach succeeds rapidly, although in some cases this procedure requires great care and dexterity. If the sound is introduced without care or with sudden force, injuries and tearing of the esophageal wall and even lacerations of the neighboring vessel occur easily, particularly in restless animals. Such cases are particularly frequent if the operation is performed by non-professional attendants who are often too ready to step in.

If the removal of the foreign body in the cervical portion of the esophagus is impossible by any method above described,

or if these methods are unpromising from the start on account of the nature of obstruction, the division of the foreign body with the aid of a tenotome may be attempted in the following manner: The foreign body is pushed to the left side, an incision is made into it with a fine-pointed tenotome, next a dull-pointed tenotome is introduced and the division of the body is attempted (Imminger). In some cases, however, esophagotomy cannot be avoided. Porcher and Morey removed a foreign body (teaspoon) from the caudal extremity of the esophagus of a dog by a laparo-gastrotomy.

**Literature.** Chapellier, Bull., 1904, 483.—Drouin, Bull., 1904, 854.—Eber, S. B., 1896, 30.—Fabretti, Vet. Jhb., 1896, 161.—Imminger, W. f. Tk., 1906, 221.—Johne, S. B., 1879, 45.—Kahn, Engelmanns Arch. f. Physiol., 1906, 355.—Maury, Rev. vét., 1899, 159.—Meltzer, Ztbl. f. Physiol., 1906, 993.—Porcher & Morey, Bull., 1898, 707.—Pr. Mil. Vb., 1903, 114.—Rolfes, Vet., 1894, 128.—Schäfer, A. f. Tk., 1896, XII, 280.—Suffran, Rev. vét., 1906, 652.—Zietschmann, S. B. 1903, 262.

## 5. Narrowing of the Esophagus. Stenosis oesophagi.

(*Verengerung des Schlundes* [German].)

**Etiology.** Stenosis of a shorter or longer section of the esophagus may be brought about by cicatricial contraction of the mucosa (*strictura oesophagi*), caused by deeply penetrating inflammations or injuries, crushing by pointed or rough foreign bodies, destruction of tissues by corroding poisons. Thickening of the mucosa or of the muscularis of the esophagus produces a similar effect, likewise an abscess or a neoplasm or other swellings, such as carcinoma, actinomycosis, papilloma, nodules due to *spiroptera sanguinolenta*. In the case of a cow seen by Joest, a pediculated, nodular, spindle-celled sarcoma of the lower end of the esophagus always receded like a ball valve during deglutition, to be again pressed into the esophagus upon the filling of the rumen. In the case of a horse reported by Schimmel a single gastrus larva, which had penetrated into the esophageal wall, produced stenosis.

Stenosis of the esophagus in the horse may be due to continuous and often repeated spasm of the cardia. The occurrence of muscular hypertrophy with dilation of the esophagus, but in the absence of any obstruction (Kitt, Fröhner and others) to which one could refer the hypertrophy and the subsequent dilatation of the esophagus, permit the conclusion that stenosis may be due to the causes given above. This is also shown by an observation of Woodruff who failed to find anything abnormal in the esophagus at the post-mortem examination of a horse which, during life, had presented the symptoms of esophageal stenosis and which, after the introduction of the finger into the cardia, had always shown strong antiperistaltic movements. Petit and Germain are, however, inclined to look upon muscular hypertrophy at the caudal end of the esophagus as depending upon chronic dilatation of the stomach, due to abnormal gas formation.



Compression of the esophagus is frequently due to neoplasms (melanoma, struma, lymphoma, myoma) exostoses, tuberculous masses in cattle, but also in dogs, tuberculous mediastinal glands, purulent or tuberculous peribronchial glands; sometimes also to arteries with an abnormal course (Labat). All these formations may cause lateral or annular compression and diminution of its lumen (*Compressio oesophagi*).

Cases of stenosis in a circumscribed place due to anomalies of development are very rare (Casparini and Serres saw such a case each in a foal, Smith in a one-month-old dog).

**Pathogenesis.** With the exception of the congenital stenosis or of cases due to spasm of the cardia, all other forms develop very slowly and the symptoms therefore rarely appear suddenly or immediately after birth, or after the weaning of the young when these are fed with vegetable food; but they make their appearance very gradually. Since inhibition of the passage of morsels of food stands in direct relation to the degree of obstruction, it may happen that a larger or firmer mass of food becomes caught accidentally in front of the obstruction. As long as arrested it will cause the symptoms of obstruction of the esophagus by a foreign body (see page 226). The convulsive contractions of the muscles of the esophagus, caused by the arrested morsel, press the latter through the narrowed place after a shorter or longer time, and deglutition is then again possible. Repeated and strong efforts at deglutition made voluntarily by the animal also assist in pressing the morsel through the stenosed portion.

**Symptoms.** The symptoms consist from the start in disturbances of deglutition. The animals begin to feed greedily, they masticate and swallow, and the mouthful first passes down perfectly normally, then, however, there are strong efforts at deglutition; the morsel may finally pass into the stomach. The efforts are repeated later at each act of swallowing or at each meal again and again, according to the degree of stenosis and to the nature of the feed, until the animal, half fed but tired out, desists from feeding. During the forced attempts at deglutition, saliva, or saliva mixed with masticated food is from time to time transported outwards through the mouth or nose in consequence of antiperistalsis. Deglutition of fluids, however, is at this time not at all or very little disturbed, but if the stenosis is quite marked, fluids will also cause disturbance. In some cases, there is only a retarded ingestion of food accompanied for some time by more or less regurgitation.

If the stenosis increases the clinical picture of complete esophageal occlusion, or more often that of dilation, gradually develops.

A considerable stenosis in ruminants leads to chronic



bloating, since belching is prevented. Bloating is frequently the first symptom in cattle (Johne) and is in certain cases, as claimed by Hamoir, not due to an obstructing impediment in the esophagus, but to pressure upon the pneumogastric nerve by tuberculous mediastinal glands. (Gruetzner, however, saw bloating of the stomach in rats only after severing the cervical portion of the pneumogastric nerve; cutting in a more caudal region had no effect.)

Some information as to the seat of a stenosis may be gained from a careful observation of the animal during feeding; energetic efforts at deglutition or a discharge of saliva mixed with particles of food from the mouth or nose appears the longer after swallowing of a morsel the nearer to the stomach a stenosis is situated. The advance of the morsel may be controlled by placing the hand upon the esophagus. The best results may be obtained by the use of the esophageal sound, but the use of this instrument is not always without danger. A sound of proper thickness, on being introduced into the esophagus, meets the impediment and its seat can be ascertained from the length of the part introduced. If now successively smaller and smaller sounds are introduced, the thinnest one, which finally passes into the stomach, gives the degree of the stenosis.

The introduction of the esophageal sound requires great care and some practice; it can, however, be easily accomplished on all animals, particularly in the horse in a standing position. The anterior end of the sound is moistened with oil, glycerine, fat or vaseline, the head of the patient is elevated and so stretched that it forms one line with the neck; the mouth is opened with a mouth gag, or if nothing better is at hand with a piece of wood or cork placed between the molars, the tongue is then depressed with the left hand which is introduced into the mouth, then the sound is grasped with the right hand, like a penholder with the anterior end somewhat raised, introduced into the mouth and pushed carefully over the left hand into the esophagus until it meets an impediment. The sound is then in place, held for a little while, is withdrawn somewhat, and again pushed forward under even, very moderate pressure. Care must be taken not to produce a perforation of the esophagus. If the sound again meets the impediment at the same place, a stenosis at this site may be diagnosticated.

In ruminants the sound is to be introduced through the central opening of a wooden crossbar used as a mouth gag (Fig. 33). (For the introduction of the sound in horses see the chapter on acute dilatation of the stomach.) This procedure, however, must never be instituted when there are signs pointing to acute inflammation of the esophagus.

**Diagnosis.** Although the disease can usually be recognized from the clinical symptoms, a definite diagnosis can only be made after the use of the sound. It is also necessary to ascertain the cause of the stenosis, and in this respect the clinical history and contributory circumstances may give information. Compression of the esophagus, due to tuberculous mediastinal glands, may, according to Johne, be recognized without fail when, aside from more or less definite symptoms of tuberculosis, there is chronic bloating without disturbance of appetite, rumination and defecation.

(see page 232). Ectasias of this type extend over a large portion of the esophagus.

Schimmel saw an ectasia in a horse with profound paralysis of the recurrent nerve which was 58 cm. long. He considered the affection to be due to *gastrophilus* larvæ which had located there while the animal was still a foal.

Basing his views on a carefully investigated case, Kelling claims that dilatation without stenosis is due to atrophy of the longitudinal muscle fibers of the esophagus, this in its turn being caused by degeneration of fibers of the pneumogastric nerve following infection.

Diverticula of the esophagus are formed rarely, and then in the following manner: A uniform dilatation has developed in consequence of esophageal stenosis or obstruction, and the accumulated masses of food have exerted pressure upon the esophageal wall; a diastasis has consequently been produced in the muscularis and a prolapse into the cleft has been formed by the mucosa, the latter again pushes against the connective tissue which yields and a diverticulum is now formed in the region of the dilation. More commonly the mucosa and the muscularis are injured by rough or pointed foreign bodies and the non-elastic cicatricial tissue which forms is subsequently dilated by morsels of food (so-called false diverticula). Genuine diverticula, which must always include the muscularis, are in some cases produced by pressure from the lumen (*diverticulum per pulsionem ortum*) by wedged-in foreign bodies, feed material, or neoplasms of the esophagus, or more rarely by traction upon the external surface by adhering heavy tumors, by contracting diseased lymph glands, between the esophagus and the trachea or a bronchus, or by the contracting cicatricial tissue following an abscess (*diverticulum per tractionem ortum*).

In exceptional cases the diverticules are formed without any obvious cause. In horses they may also be due to small, long clefts with a giving way of muscle bundles, as is seen in otherwise perfectly healthy animals (Kitt).

Storch, in connection with a case, gives it as his opinion that diverticula may be congenital and may stand in some relation to the second bronchial cleft, so that a diverticulum might be an internal incomplete bronchial cleft. Johne had previously pointed out such an explanation as possible.

Diverticula are sometimes short, sometimes they run as long parallel tubes above or laterally to the esophagus with which they may communicate by a wide opening or by a narrow cleft.

**Pathogenesis.** Food collects in the ectatic esophagus or in the diverticulum at each feeding; water likewise collects if the diverticulum is directed downwards. This finally becomes filled completely and then compresses the esophagus so that it produces stenosis or even complete obstruction. The morsels which now follow, either pass with difficulty or they become



wedged in. An ectatic place, situated in the first portion of the esophagus, is less serious if there is no stenosis back of it, since strong contractions of the muscles of the pharynx suffice to press food and drink down into the cervical portion of the esophagus.

The impaction in the ectatic portion of the esophagus creates a feeling of fullness and causes repeated convulsive contractions of the muscles immediately in front of the dilated portion and also of those within it if it contains muscle fibers.

The convulsive contractions produce a feeling of intense pain, but after more or less time they finally expel the contents of the dilated portion either into the stomach or towards the pharynx. If the latter is the case, the transport has been aided by anti-peristaltic movements. Dilatation of the esophagus in consequence of pressure more or less interferes with the proper function of neighboring organs.



Fig. 34. Ectasia of the esophagus in a horse.

### Symptoms.

The animal begins to take up food greedily; however, it de-

sists after a little while evidently satisfied, withdraws from the crib and as in obstruction or stenosis makes empty masticatory movements with lowering of the head and convulsive contractions of the muscles of the neck; smacking noises are also sometimes heard. All these symptoms are sometimes mild, sometimes intense. They are usually more marked when rough food is taken than during the ingestion of cereals or water. (In a case of Arloing a donkey showed distress only when drinking water.) With the above symptoms one sees that the patients occasionally vomit undigested food mixed with saliva, and there may be a nasal discharge of mucus mixed with particles of food. In this, as in other forms of obstruction of the esophagus, horses some-



times exhibit a mild colic. Zürn has seen periodical contractions of the esophagus running from the thoracic to the cervical portions.

By repeated attempts at deglutition the animal finally succeeds in transporting the contents of the dilated portion, either towards the stomach or towards the outside world; then the ingestion of food is again resumed. (During one period of feeding several interruptions of this kind may be noted.) The patient may in this manner take up an entire ration, although much more slowly than normally. In those milder cases where the ectatic portion temporarily becomes partially or completely empty, disturbances in the intake of water are usually seen only directly or shortly after the ingestion of solid food; they are absent or only very slight as long as the ectatic portion is empty.

Diverticula may exist without any marked disturbances of deglutition (Zürn), they may lead to only retarded ingestion of food with a discharge mixed with particles of food. The clinical picture here is similar to that of stenosis of the esophagus. The condition may become obvious only upon the occurrence of complications (aspiration pneumonia).

If a dilatation exists in the cervical portion of the esophagus, one sees a more or less circumscribed, roundish, or more cylindrical swelling in the left jugular depression, which may or may not extend toward the right side. The former condition is rather rare. The swelling is usually doughy, more rarely firm in consistency; it gives a tympanic sound before the ingestion of food, becomes larger after it, and it can be made to decrease in size by pressing or kneading; simultaneously with such manipulations there is a discharge from the nose of a slimy, eventually fetid mass of masticated food. These manipulations sometimes also cause gagging. The nutrition always suffers, generally from the start; progressive emaciation indicates some internal trouble at a time when the symptoms are not yet well marked. The more the difficulties in deglutition increase, the more the animal emaciates, and it may in fact finally starve to death.

In ruminants there is also present a chronic bloating due to the obliteration of the esophagus, which is at first temporary then continuous.

In the later course of the disease, pressure upon the trachea, the bronchi and the pneumogastric nerve after food ingestion produces disturbances of circulation and respiration; patients may occasionally suffocate in consequence of pressure (Friedberger & Fröhner) or in consequence of the aspiration of food material.

**Course.** With an increase of the stenosis behind the dilatation, or with an increasing loss of power of the muscular coat

the propulsion of the contents becomes more and more incomplete in spite of stronger efforts at deglutition. The patients cease to take food much sooner, because they become tired out earlier, and they leave more and more food untouched. When the occlusion has become complete the ingestion of food and water becomes impossible, or the wall of the ectatic portion becomes gangrenous, or it ruptures with the production of fatal ichorous inflammation.

Rupture of the dilatation or the diverticulum manifests itself in a sudden aggravation of the condition, the pulse becomes rapid, respiration forced, the temperature rises, and symptoms of ichorous inflammation of the cervical connective tissue or the pleura follow; ingested water and food can now get directly from the esophagus into the pleural cavity and with the symptoms of pleuritis there develops a horizontally rising area of dullness in both thoracic cavities or in one of them (Hora). The disturbances of deglutition may at any time produce an aspiration pneumonia with subsequent gangrene.

Dilatation of the esophagus, subsequent to obstruction, takes the same course as the latter. Such an ectasia has this in common with obstruction, that the former, like the latter, leads to swelling in the jugular depression if it is situated in the cervical portion of the esophagus.

**Prognosis.** Diffuse ectasias of the esophagus, as well as diverticula, are, as a rule, progressive affections which terminate fatally. While the prognosis is influenced by the degree of nutritive disturbance, there is always danger of perforation or of gangrene, which, of course, makes the prognosis still more unfavorable. Even if perforation should exceptionally lead only to the production of an abscess which breaks towards the outside world, the morbid changes in the wall of the esophagus and the subsequent cicatricial contractions would later on make conditions gradually worse.

**Diagnosis.** The symptoms presenting during the ingestion of food and water suggest the character of the disease, but it can be diagnosticated definitely only when we find a temporary swelling in the neck in the jugular depression which diminishes on pressure. This distinguishes the disease from inflammation of the esophagus, abscesses in the cervical connective tissue (but these may develop from an ectasia), and simple stenosis from spasm of the esophagus. Paralysis of the esophagus can be excluded by its sudden appearance and short duration. A sign of diagnostic importance is the fact that the esophageal sound can sometimes be easily pushed into the stomach, at other times, that is, if it slips into the ectatic portion or into the diverticulum, it is arrested and does not enter the stomach.



**Treatment.** Treatment can be considered only if the dilatation is situated in the cervical portion of the esophagus; in this case a narrowing may be brought about by excision of portions of the dilated wall. Collin and Schindelka have performed this surgical operation successfully. In other cases we may provide for a proper nutrition of the animals with fluid and mushy food, eliminating dry and rough feed.

(Véret in one case performed manual pressure on the cervical dilatation each time after feeding and brought about recovery within one week. Others have recommended instruments with pads which can be fastened to the neck. These could, however, be successful only in recently formed dilatations.)

**Literature.** Fröhner, Monh., 1898, IX, 349.—Güntherberg, Z. f. Vk., 1907, 492.—Kelling, S. B., 1903, 228.—Krampe, Z. f. Vk., 1907, 322.—Pr. Mil. Vb., 1896, 97.—Roloß, Pr. Mt. 1872-73, 161.—Schellenberg, Schw. A., 1892, XXXIV, 201.—Schimmel, O. M., 1904, 345.—Schindelka, Ö. Vj., 1886, 131.—Véret, Rec., 1878, 187.—Zürn, D. t. W., 505.

## 7. Neoplasms in the Esophagus. Tumores oesophagi.

Papillomata; sometimes singly in the shape of small, soft, villiferous nodules on the inner surface of the esophagus, sometimes (particularly in the middle section of the gullet) in the shape of large (the size of a fist) branched pediculated soft cauliflower masses, are comparatively frequently seen in **cattle**. Sometimes multiple actinomycomata are seen on the mucosa as prominent, flat, tough or internally softened yellowish-red nodules (Siedamgrotzky, Johnie) or single tumors are developed which may be as large as a fist (Jong).

In **horses** there are encountered in the esophagus fibroma (Dieckerhoff), melanoma (Roell, Besnard), sarcoma (Cadéac) and carcinoma (Lorenz), in **dogs** occasionally sarcoma, chondroma and retention cysts.

**Symptoms.** Tumors narrow the lumen of the esophagus in proportion to their size and consistency, and produce the symptoms of esophageal stenosis (see page 232).

One must particularly think of a tumor if there is in the left jugular depression a permanent, firm swelling, free from inflammatory symptoms, causing the sound to meet an impediment.

Treatment can only consist in the operative removal of the tumors situated in the cervical portion or, in actinomycosis, in the internal administration of iodine preparations.

## 8. Spiroptera Sanguinolenta in the Esophagus.

**Occurrence.** The occurrence of spiroptera sanguinolenta in dogs has been reported from France, Italy, China, Brazil, India, Turkestan, Japan and Tunis; 10% of the dogs of Japan and 70% of the dogs of Tunis are said by Roger to suffer from this worm.

**Etiology.** *Spiroptera sanguinolenta* is a blood-red worm 3-8 cm. long; the male is smaller than the female. The tail end of



the former is rolled up. The young forms of this worm exist in cysts contained in the abdominal cavity of cockroaches (*Blatta orientalis*).

**Natural infection** occurs from the ingestion of cockroaches, whereupon, after two weeks, small spiroptera become visible in the esophagus (Grassi).

**Pathogenesis.** The larvæ taken into the stomach with cockroaches wander back into the esophagus, bore into the mucosa and lead to the formation of hazelnut-sized or larger swellings the compartments of which contain up to twenty worms embedded in pus. Except for a small opening at the apex of the swellings, the mucosa appears intact. Some of the parasites frequently remain in the stomach, others may wander into the organs with the lymph or blood current, particularly into the wall of the aorta, where they likewise cause a local inflammation. According to Roger, the worms form metabolic products which excite the nerves of the heart.

**Symptoms.** In the more carefully studied cases intestinal disturbances were observed (painful and difficult deglutition, gagging or vomiting, empty mastication), also dry cough, dyspneic attacks, emaciation. Occasionally nervous disturbances were observed, especially weakness of the hind legs or a behavior as seen in rabies. Severe cases as a rule gradually lead to death which may also occur suddenly in consequence of rupture of the aorta.

**Treatment.** This can be only symptomatic. Better success may be had by prophylactic measures consisting in the prevention of the ingestion of cockroaches.

**Literature.** Roger, Rev. vét., 1907, 241 (Lit.).

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**Other Animal Parasites in the Esophagus.** There is found in the epithelial covering of the esophagus of cattle spiroptera scutata Mueller, also named Gongylomena scutatum, Leuckart, a filiform worm, 4-10 cm. long; its cephalic end is provided with shieldlike chitinous scales. This worm has also been found in sheep and goats; another species *G. pulchrum* is parasitic in swine.

Larvæ of *hypoderma lineata* are found during the summer and winter months in the submucous tissue of the pharynx and esophagus of cattle, where they sometimes produce a hemorrhagic infiltration.

In the intermuscular tissue of the esophagus of ruminants and horses are found very numerous sacs of psorospermiae (*Balbiana gigantea*), but they do not produce any disturbances. The same may be said of Miescher's tubules or the psorosperm sacs.

## 9. Diseases of the Crop of Fowl.

### (a) Catarrh of the Crop. Inguvitis.

(*Weicher Kropf* [German]; *inflammation ingluviale* [French]; *soft crop*.)

**Etiology.** Primary catarrh of the crop makes its appearance after the ingestion of pointed foreign bodies, of soft, scaly or, on the contrary, hard food. Richter reported numerous cases among chickens after the feeding of too dry oats. Sometimes the disease is due to the ingestion of fermenting or decaying materials (draff, distillers' mash, parts of cadavers). Too early removal of the young squabs from pigeons often causes ingluvitis in the parents, because the glands which were active during the feeding of the young birds may subsequently become inflamed.

**Secondary** ingluvitis is caused by animal parasites and is seen in thrush.

**Symptoms.** The animals lose their appetite, are listless, stretch their necks repeatedly and swallow with difficulty. The crop is tender to pressure. The fermenting contents cause a marked protrusion of the region of the crop, the latter feels soft and is tympanitic on percussion (so-called soft crop). Fetid gases escape from the beak upon pressure on the crop or are occasionally brought up by vomiting; the fluid contents are likewise emptied and they smell disagreeably sour.

These escape through the beak and partly through the nose. Disturbed nutrition leads to emaciation; the animals become very weak and frequently die. After a long continued affection of this kind or after repeated attacks a so-called pendulous crop develops frequently.

**Treatment.** One must particularly try to empty the crop of its contents by holding the animal's head down and carefully kneading the crop towards the buccal cavity. The next step consists in the administration of mild disinfectants (2% boracic acid solution, 1% sulphate of iron) or astringent solutions (alum, tannic acid, juice of lemons); peppermint tea or fennel water to which 10% hydrochloric acid has been added is also serviceable, also a 5% solution of Carlsbad salt (Klee). In very obstinate cases irrigation of the crop may be instituted with good success.

**Irrigation of the crop**, according to Klee, is performed as follows: A funnel is placed at the end of a thin rubber tube 40 cm. long; the free end of the tube is then introduced into the crop through the beak. The funnel is now filled with a 1% solution of boracic acid, it is next elevated and the solution is allowed to run into the crop. Finally the funnel is lowered and the fluid is permitted to run out. While it escapes the crop is carefully kneaded or massaged.

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The disease can sometimes be cured only by an incision into the crop. This operation also seems indicated in pendulous crop, provided the catarrhal condition has already disappeared.

Catarrh of the crop in pigeons due to the early removal of their young ones may be cured rapidly by substituting other young ones.

**Literature.** Klee, *Geflügelkrankheiten*, 1905, 76.—Zürn, *Geflügelkrankheiten*, 1882, 76.

(b) **Occlusion of the Crop. Obstructio ingluviei.**

(*Hard crop, indigestion ingluviale* [French].)

**Etiology.** Occlusion of the crop is frequently due to overfeeding, especially with dry grains (millet, corn, peas, oats) or with a feed containing much bran or chopped straw. In water fowl the disease follows upon the ingestion of certain aquatic plants (*chara*, *cynodon*, *triticum repens*, *corex*) or of *ailanthus glandulosa*. Young pheasants sometimes contract the disease after the ingestion of numerous insect larvæ. Sometimes foreign bodies (pieces of metal, stones, pieces of glass, pieces of bones, fragments of egg shells) or many small stones cause the disease, which exceptionally may be produced by animal parasites (Railliet and Lucet).

**Symptoms.** Obstruction of the crop manifests itself by the fact that the animal becomes listless, it repeatedly opens the beak and the latter sometimes discharges an ill-smelling fluid. The appetite is absent either from the start, or after some time if the obstruction is due to a foreign body. The crop appears enlarged; its wall is tense, elastic, sometimes doughy, sometimes hard in consistency (so-called hard crop), depending upon the mass of accumulated food and upon the amount of gases that have formed. Foreign bodies in the crop may be felt through its wall or may be demonstrated by a Roentgenograph.

**Course.** Obstruction of the crop rarely disappears spontaneously. If nothing is done, chickens usually die of exhaustion after a few days; water fowl, however, succumb to suffocation after a few hours (Dupont). This difference in the course can perhaps be explained by the fact that the crop of chickens is really an extrusion directed outward, while the crop of water fowl is a circular dilation of the esophagus, which may easily compress the trachea. Occasionally rupture or perforation of the crop occurs. Rivolta and Delprato saw the formation of an enormous crop (so-called pendulous crop) in a hen (Kitt).

**Treatment.** If the contents are not too hard, massage of the crop, and kneading of the contents towards the beak, are in-

dicated. If the crop has become hard or if the massage has been unsuccessful, an incision into the crop must be performed.

**Incision into the crop** is comparatively simple and without danger except in pigeons. The feathers are removed with scissors in a region about two fingers wide and one finger long. Then one makes a pointed incision in the middle of the crop and prolongs the wound parallel with the long axis of the neck from 1 to 1½ cm. If the contents of the crop consist of small pieces they can now be removed by kneading; if larger leaves are present they can be removed by the aid of a pair of forceps. The wound is then irrigated and sutured. It usually heals rapidly and the stitches can be removed in five to seven days. On the first day after the operation the animal must not receive anything except stale white bread soaked in claret; after thirty-six hours abundant water, but only a small amount of soft food.

**Literature.** Dupont, Dict., 1874, X, 223.—Klee, Geflügelkrkh., 1905, 78.—Railliet & Lucet, Rec., 1890.—Richter, Dresd., Ber., 1908, 254.—Zietschmann, S. B., 1903, 264.—Zürn, Geflügelkrkh., 1882, 173.

### (c) Animal Parasites in the Crop.

Several species of thread worms occur in the esophagus, crop and stomach of fowls; some of these may cause catarrh of the crop and numerous cases of death. In the crop and stomach of chickens are sometimes found many individuals of **Dispharagus nasutus** (*Filaria nasuta*) **D. spiralis** and **D. laticeps**. In spite of good appetite, the infested animals become much emaciated and finally die. **Trichosoma contortum** living in the crop of ducks and geese, produces a severe catarrh and subsequent dilatation of the crop (Railliet & Lucet), also frequently rapid emaciation, drowsiness, sometimes epileptiform convulsions; after five to ten days, symptoms of obstruction of the crop and of catarrh follow. In order to make a diagnosis, the procedure consists in irrigation of the crop and macroscopic and microscopic examination of the material which is washed out.

Treatment is not successful. Animals may be protected prophylactically by keeping them away from contaminated water.

**Literature.** Klee, Geflügelkrkh., 1905, 80.—Railliet & Lucet, Rec., 1890, 13.



## SECTION V.

### DISEASES OF THE STOMACH AND INTESTINES.

#### 1. Vomiting. Vomitus.

(*Erbrechen* [German]; *Emesis*.)

**Etiology.** Vomiting is produced by an irritation in the vomiting center of the central nervous system, the stimulus acting upon certain parts of the nervous system directly or by reflex irritation from a spot in the gastro-intestinal tract or other portion of the body. Hence vomiting may be part of the clinical picture of a variety of diseases, but it is most frequent in the course of certain diseases of the stomach or of the small intestines.

One of the most frequent causes of vomiting is overloading of the stomach. For this reason vomiting very frequently occurs among dogs and swine, but it is not rare in ruminants (in consequence of overfilling of the paunch and of bloating) and also occurs in horses. Irritation of the gastric mucosa is the cause of vomiting if the latter occurs after the ingestion of irritant acrid feed (cattle sometimes vomit after the ingestion of alfalfa), after the administration of acrid or irritant medicines (emetics) and also during catarrhal or inflammatory diseases of the gastric mucosa (distemper, parasites in the stomach, hog erysipelas, ulcer, etc.).

Deli observed in one community an enzootic occurrence of vomiting among horses; both at rest and in motion the animals vomited repeatedly a watery fluid. According to the opinion of the author, the ingestion of muddy water had caused a relaxation of the cardia with subsequent mild vomiting.

Of the various diseases of the intestines, obstruction of the intestines especially leads to vomiting. In such a case antiperistalsis often occurs, which propels the contents of the intestines toward the stomach; the vomitus, therefore, is mixed with intestinal contents. In displacements of the intestines and in acute peritonitis the emetic stimulus may originate in the peritoneum.

That the internal genital organs may furnish the exciting stimulus for vomiting has never been shown in veterinary science. Irritation of the buccal mucosa occasionally also

causes vomiting. For this reason vomiting is not infrequently observed in carnivora and omnivora during the course of stomatitis, after the lodgment of foreign bodies in the pharynx (dogs often vomit after the ingestion of grass) and also during convulsive attacks of cough. Finally, vomiting is often seen in severe primary or secondary pharyngitis.

Vomiting is of central origin (the stimulus originating in the central nervous system) when it occurs with injury or disease of the brain or during transportation in ships (seasickness). A similar origin is probably responsible for the occasional vomiting of uremia or cholemia.

There are certain chemicals which cause vomiting by irritation of the emetic center (veratrine, apomorphine, chloroform). Hogs, dogs, cats are the domestic animals which vomit easily and frequently; ruminants and rabbits with more difficulty and more rarely; horses quite rarely and only with great difficulty in their attempt to expel the contents of the stomach. Vomiting in horses is therefore of much greater significance than in the other species of animals named. The differences as to the various species are principally due to a lesser irritability of the emetic center of the herbivora and especially of the horse when compared with other animals. The rarity of gagging, which is parallel with the rarity of vomiting, likewise points to this cause. The unfavorable anatomical conditions in the stomach of the horse must act against the expulsion of the contents of the stomach; but they cannot explain the absence of the characteristic reflex motion, the gagging. That the influence of anatomical conditions is much overestimated appears from the observation that in acute dilatation of the stomach horses belch a good deal but rarely gag and vomit. (Forssell claims that a plug of mucosa in the region of the cardia closes the latter in consequence of the displacement of mucosa on the loose submucosa.)

**Symptoms.** Hogs, dogs and cats vomit without any particular efforts. After a certain degree of unrest, the head is stretched, lowered to the floor, and the mouth then discharges part of the gastric contents, particles of food mixed with mucus, occasionally also with bile or fecal matter. In cattle vomiting is preceded by greater restlessness and greater efforts; the animal pulls back from the manger, the fore-legs tremble, the hind legs are placed under the abdomen, the head is stretched out, the mouth then expels after a short inspiration a large amount, often more than ten quarts of fluid or of food. Horses make similar and still more noticeable efforts; their whole deportment betrays much excitement, the neck, the muscles of which are very tense, is bent forward, the head is bent towards the thorax, whereupon the vomited matter is mostly expelled through the anterior nares. Vomiting is observed either as a single act or it is repeated several times or it may last for hours.



behavior as in genuine vomiting, but no gastric contents are expelled.

Belching or eructation is a normal occurrence in ruminants. It is however also and more frequently observed under morbid conditions; then the expelled gases frequently have a repulsive odor. In other animals belching is always an indication of abnormal gastric fermentation.

The significance of vomiting depends, of course, upon the cause and upon the underlying disease. It is always gravest in horses where it occurs in the course of dangerous diseases of the stomach (excessive dilatation of the stomach, severe gastritis, partial rupture of the wall of the stomach, etc.), or through an excessive irritation of the gastric nerves; in horses vomiting also gives rise to an aspiration pneumonia with comparative frequency; this is much rarer after vomiting in other animals. Exceptionally an animal may suffocate during vomiting, because the expelled feed masses may get into and obliterate the larynx or the nasal cavity (Sequens, Zschokke).

Older authors have looked upon vomiting in the horse as a sign of rupture of the stomach. It is however settled beyond doubt that these animals cannot vomit any more in total rupture of the stomach, because the gastric contents are expelled into the peritoneal cavity in consequence of the contractions of the wall of the stomach and of the abdominal press. Still, in partial rupture of the wall of the stomach an intense emetic irritation may cause vomiting. Partial ruptures are occasionally found in the stomach of horses which have vomited some days before death (from other diseases) had occurred. Rupture of the stomach may become larger or even complete after vomiting has occurred. That horses are able to vomit even if the stomach is absolutely intact has been shown convincingly by post-mortem examinations preceded by vomiting during the life of the animal.

Emetic irritation also causes a reflex relaxation of the cardia and of the esophagus, and this the authors have frequently seen upon the introduction of the stomach tube. Relaxation of the cardia may also be due to a degeneration and to a tear between muscle bundles.

**Treatment.** Except in horses, vomiting after overloading of the stomach and after poisoning is beneficial because the stomach becomes relieved or the poisons are removed. In such cases, therefore, it is not best to stop vomiting but this should be aided by the internal administration or subcutaneous injection of medicines (apomorphine, veratrine, ipecacuanha), or it may even be produced artificially, the emetic irritation being stopped only after too frequent attacks have become distressing. Vomiting due to any other cause, however, should be stopped because it distresses the animal and disturbs its nutrition. Symptomatic treatment consists in the application of cold (swallowing pieces of ice, cold applications in the gastric region in small animals, narcotic medicines, opium internally, chloral hydrate or bromides per os or per rectum in the form of enemas or suppositories, morphine subcutaneously). Vomiting in dogs may sometimes be stopped with black coffee (one wine glass full to a cup full).

Holterbach arrested very obstinate vomiting in a dog by the administration of yohimbinum muriaticum Spiegel one tablet of 1 mgm. every two hours.



animals are most commonly attacked, more rarely cattle on pasture.

**Etiology.** The affection is usually due to the ingestion of unusually large masses of feed; it is immaterial whether the food is of the highest quality or unsuitable in character. If the animals obtain palatable hay, green feed, flour or grain feed without restriction, or if they have broken loose, they are liable to overfeed, particularly if they had been starved for some time. (There are some countries in which it is customary to increase the live weight of animals to be sold for slaughtering by feeding them excessive quantities of feed before the transfer is made.) Animals occasionally overfeed on rich pastures, particularly if they have been fed sparingly in the barn. The affection is also brought about by feeding with voluminous feed which is too dry or improperly prepared (chaff, straw, coarse clover, hay, etc.); a low nutritive value causes the animals to ingest great quantities. Beets, leaves of beets and potatoes, particularly in a boiled mushy condition, kitchen garbage, distillers' or brewers' mash, mouldy or frozen feed may likewise cause dilatation of the rumen. Finally, lack of water (due to neglect) during dry feeding, becomes responsible for the disease.

Predisposing factors sometimes act as contributory causes, such as physical weakness, continuous stay in the stable, ingestion of too cold water, prevention of mastication by overexertion after feeding, sudden change from green to dry feeding.

Cadéac holds that dilatation of the rumen as well as acute bloating of ruminants always follows a preliminary paresis of the rumen, so that the former are always secondary conditions. Numerous observations however show that an excessive ingestion of food may bring about the affection in animals with a rumen functioning absolutely normally. One is justified in drawing the conclusion that a normally developed and normally functioning muscularis of the rumen cannot be stretched beyond a reasonable limit.

There is, however, no doubt that the rumens of different individuals differ as to the power of its muscularis, and that external conditions may exert a good deal of influence.

Eber looks upon dilatation of the rumen as a type of idiopathic paresis of the rumen in contradistinction to sympathetic paresis of the rumen due to organic affections of the different parts of the stomach and of the neighboring organs.

**Pathogenesis.** After the effects of one of the enumerated etiologic factors have become manifest, there exists an improper relation between the mass and weight of the ingested food and the ability of the rumen to the detriment of the latter. The consequence is that the masses of feed present in the rumen are not properly mixed and moved, or not at all, in spite of the fact that the muscularis is stimulated to powerful contractions. The contents of the rumen then swell more or less and develop, in consequence of fermentative processes, an abundance of gas. The more juicy the feed has been, the more rapidly and the more abundantly gas forms. If the feed has been too dry, gas is not formed at all, or a small amount only is formed



after some time. The increasing tension of the wall of the rumen causes an increase in the power of contractions, which is painful to the animal. In milder cases the powerful muscular contractions succeed in transporting a portion of the contents of the rumen, and this relieves the morbid condition. In excessive overloading, the contents do not move, however, in spite of the energetic muscular contractions, and even the escape of gases becomes impossible. After some time there occurs then a paresis of the overworked muscles of the rumen. The more, rapidly the rumen becomes dilated by masses of swollen food and gases the more respiration and circulation become interfered with. Decomposition of the contents of the rumen occasionally produces poisonous or irritating substances, which may cause either a local inflammation or a general intoxication.

**Anatomical Changes.** The abdomen appears greatly dilated, the diaphragm is pressed forward, the much dilated rumen contains excessively large quantities of repulsively smelling feed in addition to variable amounts of gas. On post-mortem examination one finds the signs of suffocation. If the disease has lasted somewhat longer, the omasum contains desiccated masses of feed; the mucosa of the rumen, sometimes also that of the other parts of the stomach, shows diffuse reddening or hemorrhagic points.

**Symptoms.** The animal refuses its food; if in the open air they do not move, they place the legs under the abdomen or spread them apart, arch their back and stare. They manifest colicky pains and move the head towards the abdomen and push the latter from time to time with their horns, or they kick with their hind legs, wag their tails, groan, lie down occasionally but get up again at once. The colicky pains, while not attaining a high degree, are rarely absent. Groaning is usually heard, either only during a change of position of the body (getting up, lying down) or independently of it.

Appetite and rumination are only diminished in the milder cases but are absent from the start in the more severe type. Even in milder cases complete lack of appetite supervenes if the animals are offered a chance to ingest rough feed. The ingestion of water ceases first, although thirst seems to be increased in some cases. Occasionally the patients execute masticatory movements and foamy saliva runs out of the mouth. Repulsively smelling gases are expelled by belching, from time to time. Occasionally there occurs gagging, even vomiting, with the expulsion of much feed mash.

The abdomen is at first only moderately enlarged and the depression of the left flank seen on an empty stomach is filled out. In the further course or in excessive overfilling, the size of the abdomen is much increased, particularly on the left side,



and the left flank depression has been changed into a distension. The rumen feels doughy, firm or even hard as a stone. The same impression is created by a rectal exploration of the rumen; frequently this method makes the existing conditions much clearer than external palpation (Gebauer). The animals try to avoid firm pressure upon the rumen. At this time percussion of the left flank gives a dull and empty sound. The picture changes after the ingestion of a more or less fermentable feed, because then the left depression feels elastic on slight pressure and gives a tympanitic sound on percussion. A firmer pressure, however, usually suffices in these cases to reveal the presence of firm masses of feed under the layer of gas. The movements of the rumen are infrequent, slow and sluggish, or are entirely absent. The same is true as to the rumen sounds.

Incompleteness or absence of the rumen movements or sounds does in itself not prove that the muscularis does not contract, or contracts less energetically. The movements of the rumen can become visible, and the sounds audible only when the contents of the rumen are propelled.

Defecation at first takes place at normal intervals; later an increasing constipation develops. There may exceptionally be diarrhea in dilatation of the rumen due to juicy feed (leaves of beets, beets).

Respiration is at first only moderately accelerated and superficial; as meteorism develops secondarily, it becomes more and more forced. The pulse beat is influenced in an identical manner. The secretion of the milk in cows becomes much diminished.

Occasionally weakness of the hind legs with staggering gait, inability to stand up, and even extension of the parietic weakness to the anterior part of the body or a picture similar to parturient paresis has been seen in cattle and sheep. (Schmidt, Pröger, Born).

**Course.** The disease may make its appearance either immediately after the ingestion of food, or several, up to twelve hours later, and then it slowly reaches its climax, the time elapsing after ingestion depending upon the nature of the particular food. After ingestion of easily fermentable feed, development may be as rapid as in acute meteorism. The disease usually terminates in recovery. The very mild cases frequently end in recovery on the first or second day and without any treatment whatever. In severe cases, even under the proper diet, the animals may be sick for three to ten days or even longer. Severe cases end only exceptionally in recovery in a short time, namely, when the contents of the rumen are diminished considerably in consequence of vomiting. Recovery is indicated by the return of appetite, rumination, and by the visible movements of the rumen, frequent belching and abundant defecation. If properly and timely treated, the disease rarely takes a fatal course, nevertheless many fatalities have been observed in



sheep. Death may occur in consequence of severe meteorism in the second half of the first day of sickness; in sheep even with apoplectic rapidity. If the disease has been caused by the ingestion of dry or heavy food, or of food containing abundant flour or boiled potatoes, then the animals die only after the course of several days, or even some weeks, from inanition or gastro-intestinal inflammation, which is announced by elevation of temperature, rapid and weak pulse, clouding of the sensory organs and general weakness.

**Diagnosis.** The sudden appearance shortly after a rich meal, the enlargement of the abdomen, especially in the lower portion of the left side, the firm consistency of the rumen and considerable dulness on percussion in this region, are symptoms characteristic of the disease and distinguish it from the more fulminant development of acute meteorism. In the latter, atony of the fore-stomachs develops gradually, reveals an intermittent course without colicky pain and without stuffing of the rumen with masses of feed. Traumatic gastritis sometimes starts with symptoms similar to those of dilatation of the rumen, but it may be differentiated from the latter by the clinical history, the tenderness of the region of the xyphoid cartilage, the obstinate character of the disease and eventually by the administration of eseridine or veratrine in connection with arecoline (see foreign bodies in the fore-stomachs). Other inflammations of the gastro-intestinal tract run a febrile course in contrast to dilatation of the rumen and lack the filling of the rumen with doughy masses. Compression of the intestines, intussusception or torsion of the uterus are similar to dilatation of the rumen on superficial observation only, because the ingestion of food and rumination cease suddenly and there are colicky pains. However, a different history is obtained; there is no stuffing of the rumen and there is marked restlessness. Moreover, rectal and vaginal examination (spiral folds) show the distinguishing features.

**Prognosis.** This depends upon the degree and duration of the disease, but the nature of the ingested food must also be taken into consideration. The more fermentable the food or, on the contrary, the more dry it is, the longer the duration and the more unfavorable in general the prognosis. Complete recovery can, as a rule, not be looked for if the disease has existed six to ten days without any sign of improvement (Eisele). During a protracted course of the disease fatal complications arise frequently (gastro-intestinal inflammation or peritonitis), or chronic atony of the fore-stomachs remains as a sequel.

**Treatment.** Complete withdrawal of rough feed for one or two days is indicated before everything else, an improvised muzzle being used to prevent the ingestion of straw (Eber). An-



other important step is massage of the rumen to assist in or directly bring about the movements of the rumen. The effect of massage can be increased by methodical leading around of the animal and by repeated frictions of the regions of the flanks and sides.

**Massage of the rumen** is to be carried out by making moderate pressure with both fists along the whole left flank, both upward and downward, and also boring motions upon the contents of the rumen. Massage is kept up for five to ten minutes and, except in the case of sows near parturition, it is followed by the same procedure on the right side. When the rumen is very much overfilled, massage must be carried out simultaneously on both sides by two persons. The procedure is repeated every two to three hours; in milder cases, however, only two to three times a day (Eber, Schlamp).

Cases of dilatation of the rumen of moderate severity may be cured by the withdrawal of rough feed and by a systematic massage. The latter can be supported effectively by certain laxatives and these should be administered, particularly in the severer cases. Best suited are those remedies which can be injected subcutaneously, for instance, eseridinum tartaricum (eseridine 0.2-0.3 gm., acid tart. 0.01 gm., aquae dest. 20 cc. for adult cattle [Eber]), veratrinum sulfruricum (0.01-0.15 gm. for adult cattle; 0.03-0.04 gm. for goats and sheep, dissolved in fifty parts of alcohol), eserine (0.15-0.20 gm. for cattle [Feser]); the latter, however, sometimes causes excitement, dyspnea, abortion or even suffocation, and should not be used in small ruminants. Pilocarpine (0.3-0.5 gm.) may be used in cattle, without hesitation (Kunke). Tartar-emetic (3-4 gm. daily per mouth—great care in the administration), chloride of barium (6-10 gm. in a quart of water per mouth) and veratrum (10-20 gm. of the powdered root or of the tincture) may also be serviceable. These medicines are also indicated in milder cases in place of massage of the rumen, when the latter for some cause or other cannot be carried out. The internal administration of other medicines appears superfluous, except, perhaps, hydrochloric acid and bicarbonate of potash with equal parts of common salt; these may be given to stimulate the appetite after the removal of the masses from the rumen has begun (Eber).

If there is danger of suffocation, puncture of the rumen will be of service only if there is a strong development of gas; otherwise it only causes a moderate transitory improvement. In the latter case rumenotomy cannot be avoided in order to remove the food mass through the wall of the rumen.

Following the operation the grave symptoms decrease at once, the animal becomes more lively, moves more easily, rumination and defecation appear and digestion becomes normal within twenty-four to forty-eight hours.

Since the contents of the rumen as a rule are matted together and are strongly fermenting, it is advisable to wash out the organ with a dilute non-corrosive disinfectant solution through

the opening made after the contents have been removed. For the next few days the animals should receive only a scanty diet of palatable and juicy tubers or fresh forage.

**Section of the rumen** is made at the same place where puncture is usually made. A pointed knife is inserted into the rumen and an incision 7 to 8 cm. long is then made in a vertical direction through all layers of the abdominal wall. Since the margins of the incision into the rumen will later on glide away from the wound of the abdominal wall and since therefore the contents of the rumen could subsequently get into the peritoneal cavity, it is well to unite the margins of the incision into the rumen with those of the abdominal wall by a baseball suture. As a matter of fact it is still better first to incise only the abdominal wall, then to suture the rumen to the margins of the abdominal incision and finally make an incision into the rumen within the oval space that has been united with the abdominal wall.

After opening the rumen gases escape with considerable force, then the food follows in quantities dependent on the amount of internal pressure and the consistency of the contents. Enough feed is sometimes expelled that the abdomen at once assumes its normal configuration. If, however, the food material has been rather dry, it must be removed, often two to three buckets full, by the hand introduced through the wound. (Brognier has constructed a forceps-like instrument with spoons for the removal, but this appears entirely superfluous.)

The incision wound usually heals promptly, and complete recovery is of course hastened by proper closure of the incision and proper aftertreatment.

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### (b) Acute Bloating of Ruminants. *Meteorismus acutus ruminantium.*

(*Akutes Aufblähen der Wiederkäuer, Trommelsucht, Blähsucht, Auflauf* [German]; *Tympanitis, Indigestion gazeuse ou méphitique, météorisation aiguë* [French].)

Acute bloating of ruminants is a morbid condition with rapid dilatation of the rumen and reticulum in consequence of rapidly-forming gases.

**Occurrence.** Acute bloating of ruminants is seen frequently and particularly during the warm season and in animals on the pasture. Cattle and sheep are affected most commonly, goats more rarely. The disease appears often simultaneously in several or many animals of the same herd or of the same neighborhood.

**Etiology.** The cause of acute bloating is the abundant ingestion of easily fermenting food. Most dangerous in this respect are leguminosæ (clover, alfalfa, esparsette, vetches, peas, lentils, buckwheat) before they have flowered, i. e., at a time when they contain much water and are fine in texture. However, other green foods (young field plants, luxuriant grass, leaves of cabbage, beets, and of young potatoes; in sheep also cereals germinating in stubble fields); vegetable bulbs (beets



and potatoes) also germinated malt, waste of sugar beets, draff, are liable to cause acute bloating if the animals have ingested large amounts of such substances without much dry food.

In sucking or weaned calves the disease sometimes occurs after the ingestion of unusually large amounts of milk; when the latter has coagulated rapidly, it is not very accessible to the gastric juice and undergoes abnormal fermentation.

While all these food stuffs may cause acute bloating, they vary considerably as to their dangerous nature. According to general opinion, clover, particularly red clover, is most dangerous; but even the same food material varies. The more luxuriant and the more juicy the food, the more easily it causes acute bloating. The greatest prevalence of acute bloating occurs in deeply-situated valleys with rich soil, and in years when the spring has been cold, and when the plants, which had been backward for some time, grow the more luxuriantly when warm weather sets in. The same circumstances cause bloating after pasturing on fresh stubble fields where weeds have been exposed to the sun a short time only and where dropped-off grains have germinated. The dangerous nature of feed plants becomes increased when they are strongly moist with dew or frosted or frozen or wet with rain. Bloating, therefore, is generally more common during pasturing in the early morning, during cool, moist weather, in spring and in fall. The danger is further increased by watering immediately after pasturing or after feeding. A withered green feed or green feed heated in consequence of storing it in large masses also causes bloating more easily than freshly-cut feed. This explains the frequent occurrence of bloating on holidays during stable feeding. Potatoes, beets and bulbous plants are more dangerous after decay has begun than when they are fed fresh. It is also claimed that bloating is easily brought about when animals swallow a lot of air while pasturing against the wind.

Predisposition to acute bloating varies as to the individual susceptibility. Greedy feeding predisposes to the affection, hence we see it frequently in starved animals and in animals permanently or generally stabled, when they are driven to a dangerous pasture. The variable efficiency of the muscularis of the rumen also plays a rôle. Physical debility, convalescence from various diseases during permanent stable feeding cause the movements of the rumen to be less energetic than under more favorable circumstances; the same occurs from violent motion or physical effort directly after the ingestion of food. Adhesions of the rumen may completely interfere with the movements of the organ.

(Cadéac holds that both dilatation of the rumen and acute bloating are conditions secondary to atony of the muscularis of the rumen.)

Bloating occurs as an affection secondary to the ingestion of certain poisonous plants (spotted hemlock, water hemlock,

species of *ranunculus*, *colchicum autumnale*, tobacco, mould) which produce a paralyzing effect through their poisons upon the muscularis of the rumen. This form of bloating does not usually attain a very high degree. Obstruction of the esophagus causes severe meteorism, particularly if the food previously ingested is fermentable.

**Pathogenesis.** In consequence of the rapid formation of gas the rumen becomes dilated in a short time, and the disturbances are similar to those caused by overfilling of the rumen combined with meteorism (see page 250), but with this difference that bloating occurs much more rapidly and the symptoms are more fulminant, occasionally even leading to rupture of the rumen.

**Anatomical Changes.** A considerable increase of the abdomen and of the rumen becomes at once manifest. The wall of the rumen is so tense that it can hardly be pressed in at all with the hand. Sometimes, one may be able to recognize during life a rupture of the rumen or of the diaphragm. On post-mortem the lungs are compressed by the diaphragm bulging into the thorax; they are dark red and full of blood. The vessels outside of the abdomen, especially the veins and particularly those of the subcutaneous connective tissue, are filled to their utmost with black red, possibly uncoagulated, blood. The signs of death from suffocation also include punctate and striate hemorrhages into the serous membranes, especially into the pleura and pericardium.

On post-mortem examination the rumen is found to contain soft, mashy, fermenting food masses and large amounts of gases which are inflammable and burn with a blue flame.

The composition of the gaseous contents of the rumen varies according to the type of food and to the nature of the fermentative process. Carbonic acid is always present to a large extent (50 to 80%); the amount is the larger the more fermentable the feed has been and the larger were the morsels upon ingestion. Next comes methane gas ( $\text{CH}_4$ ) which makes up 16 to 39% of the gases. There are also present sulphuretted hydrogen, nitrogen and oxygen, the latter two derived from the air which has been swallowed. Oxygen, however, may be used up completely during the fermentative process. In starving animals the amount of carbonic acid becomes smaller, while methane, nitrogen and oxygen are increased (Lungwitz).

**Symptoms.** The animals cease to feed, stand motionless, place their feet under the abdomen or spread them, curve their back, turn their head around towards the abdomen, and stop ruminating.

A short time later an increase in the circumference of the abdomen becomes noticeable; it soon reaches a high degree. The increase takes place all around the abdomen, becoming, however, most marked on the left side, particularly before death. The flanks, especially the left one, become flattened; in severe cases the left flank may so protrude that its apex is higher than



the back. The abdominal walls are very tense, can be pressed in only with difficulty, and at once protrude again after removal of the pressure. The left hand placed over the region of the rumen cannot feel any movement of the latter. Percussion over a large territory, sometimes down to the lower third of the abdomen, is of low pitch, excessively full, not tympanitic and occasionally metallic, in the region of the left flank. As bloating progresses, and as the walls of the abdomen and of the rumen become more tense, the percussion sound becomes weaker, without, however, becoming very weak. The rumen sounds (friction) are no longer heard; crepitation, however, becomes more distinct.

Occasionally gases are expelled from the rumen and vomiting may exceptionally occur; defecation occurs in the beginning, but becomes impossible later on.

As the abdomen enlarges, respiration becomes more and more difficult. The animals attempt to move especially the middle ribs to a higher degree, they dilate their nostrils, open the mouth and stretch out the tongue.

The mucous membranes soon assume a bluish-red color, their vessels become dilated and form a well-marked reticulum, particularly in the conjunctivæ; the veins of the subcutaneous connective tissue (on the neck, on the udder) likewise become dilated. The pulse is accelerated in proportion as the abdomen is more and more distended; the pulse then becomes gradually weaker and finally can no longer be felt.

In the further course of the affection the animals move restlessly about, expressing intense anxiety, and perspiration appears on the skin. Foamy saliva flows from the mouth. The region of the back and croup sometimes develops a cutaneous emphysema (Reynal, Woehner).

In very severe cases the general cyanosis gradually increases, the heart beat becomes bounding, the extremities and the ears are cold, the animals stagger, break down and finally die with convulsions.

**Course.** After the ingestion of large masses of very rapidly fermenting feed, gas is formed very quickly, even during digestion or immediately after it, and the gas formation progresses so rapidly that suffocation may occur within a few hours. In other cases the development of gas is slower, powerful convulsive contractions of the rumen, and frequent belching in combination with vomiting preventing the excessive accumulation of gas; in the meantime the fermentation itself becomes less and the animals recover completely.

**Diagnosis.** Rapid increase in the size of the abdomen in connection with strong tension of the abdominal wall, increase in the intensity of the percussion sounds over a large area, give a characteristic clinical picture. The behavior of the sound on



percussion and the changes in the left flank distinguish the disease from overfilling (dilatation) of the rumen; chronic bloating and atony of the fore-stomachs are much slower in development and their symptoms occur in frequently repeated attacks. Bloating due to obstruction of the esophagus or of the cardia is diagnosticated on the basis of a different history and in the former the sound encounters an impediment; belching is also absent. It must not be forgotten that gastritis due to foreign bodies sometimes is initiated by acute bloating.

**Prognosis.** Acute bloating is usually much more dangerous in sheep than in cattle, evidently for the reason that in the comparatively small body of the sheep a minor excess in gases may bring about serious disturbances of respiration and circulation and that the comparatively weak muscularis of the rumen is unable to overcome the pressure and expel the gases. The prognosis (aside from those measures which may bring about recovery) is the more unfavorable the larger the amount of ingested food and the more fermentable it is. Everything else being equal, bloating caused by clover is most dangerous, because a fulminant course brings about an intimate admixture of the food contents and the gases. The course of the affection is also unfavorably influenced by debility, early youth or advanced age and preceding disturbances of nutrition. The more animals get sick simultaneously, the more losses are to be expected, because the smaller is the possibility of timely treatment.

**Treatment.** Various methods are employed to remove the gases from the rumen; but those are primarily to be employed which will permit the escape through the natural route, i. e., through the esophagus and the mouth, the operative production of an artificial opening being indicated only after the other means have failed or in the immediate presence of danger of death. Massage of the flank must be practiced on both flanks while the foreparts are elevated (page 253); according to Eber, this procedure is the most reliable and simplest method to remove bloating in cattle, except when bloating has been produced by obstruction of the esophagus. Compressing the abdomen by tying a rope around it or a constant, strong, even pressure on the left flank, may also bring about good results (Rychner).

Elevating the animal serves the purpose to bring the gastric entrance of the esophagus in a position above the masses of feed, and this may be accomplished in the following manner: A stable door or a strong board is laid obliquely over a box, barrel or bundle of straw. The animal is then placed with its front legs on the improvised inclined plane and supported in this position by two persons who get a hold of the thorax. In an emergency the front feet of the animal may be placed in the crib (Dommerheld). Some kind of an arrangement also must be improvised if the animal is on pasture. Smaller animals like sheep may be placed on their hind legs.

The effect of driving up hill is similar to that of massage of the rumen with the animal raised in its foreparts, and it is,

therefore, often made use of with good results in mountainous countries (Eber). Energetic contractions of the rumen may also be produced by freely soaking the flanks with cold water, or by driving the animals into a river (the latter method is to be recommended if numerous cases of the affection occur simultaneously in sheep).

The effect of massage of the rumen is materially assisted by such measures as will cause belching or vomiting. A very simple and frequently effective method consists in irritating the velum and the pillars of the fauces of the animal with a dull, flexible rod (the blunt end of a whip wrapped with cloth); while this is being done the mouth must be forced open and the tongue drawn out. This frequently causes a good deal of belching with the expulsion of very large amounts of gas, sometimes mixed with food particles. It is advisable to stand on the side of the head of the animal, so that the very fetid gases do not get directly into the face of the practitioner. It is less satisfactory to merely pull out the tongue of the animal, moving it up and down in the mouth with a rope provided with knots or made of straw, or to apply a straw rope soaked in tar or other nauseating substances to the mouth of the animal.

The removal of the gases in the rumen through an artificial opening is only indicated if the condition of the patient becomes worse in spite of all other means, if suffocation is threatening, or when the animals are on the floor, so that the common methods of treatment have become impossible. The introduction of the stomach tube is not a very dangerous procedure; but it is usually not successful in primary bloating, since the inner opening of the esophagus lies behind the masses of food, so that the tube will immediately become obstructed by food particles. This defect may be remedied by an elevation of the foreparts of the animal; however, when this is done, massage is usually sufficient and makes the use of the stomach tube superfluous (Eber). If the dyspnea is severe, the introduction of the stomach tube may be injurious and may lead to fatal asphyxiation.

Monroe's stomach tube consists of a spiral wire covered with leather, or of vulcanized rubber or guttapercha, and terminates at one end in a blunt knob with several openings. It is introduced in a manner previously described (see page 233), and after its internal end has entered the rumen the expulsion of gases may be supported by pressure upon the left flank.—Sendrail recently recommended a tube formed by a spiral metal band which is said to be much more durable than the old style. The stomach tube for horses is perhaps most serviceable, because its wide lumen and smooth wall may permit the expulsion of particles of food more easily than the older instruments.

If immediate danger has been removed by one of the methods recommended, the animals must be kept under further observation so that a repeated accumulation of gases can be met by proper means.

Puncture of the rumen must be reserved for cases of primary meteorism after the other methods have failed and when

there is immediate danger of death. In some cases puncture of the rumen, particularly if performed by laymen, may cause a more or less extensive peritonitis or the adhesion of the rumen to the abdominal wall, which will subsequently endanger the health of the animal, more or less.

**Puncture of the rumen** is performed with a long trochar held in the closed hand; it is pushed (contained in its protecting tube) into the most prominent point of the left flank or into the center of a line which connects the external angle of the ileum with the middle portion of the last rib. An incision into the skin may have been made previously. The point of the trochar is directed towards the right elbow. After penetration the stiletto is withdrawn and it or a thin rod is used to remove particles of food which may collect in the tube of the trochar. In order to prevent too sudden congestion of the abdominal vessels or rupture of vessels, or anemia of the brain, it is advisable to let the gases escape gradually. The tube of the trochar should then be closed with a cork and left in place for several hours, safely secured by a bandage wound around the rump of the animal.

In an emergency, puncture of the rumen may be performed with a strong pointed knife. The latter, first carefully cleaned, is held with its blade towards the front and is pushed 8 to 10 cm. deep into the rumen; it is then rotated at a right angle in order to produce a gaping wound.

The punctured wound usually heals rapidly, but a wound which has been made with the knife should be closed by two or three sutures.

The internal administration of drugs plays a very subordinate rôle in the treatment of primary meteorism and even so-called neutralizing agents (ammonia, lime water, potash) will not materially diminish the amount of gases contained in the rumen. However, the subcutaneous injection of medicines which stimulate the movements of the rumen may in some cases be indicated (see page 254). A moderately advantageous effect might also be expected from agencies liable to inhibit fermentation. Among these may be mentioned subsulphate of sodium (100.0-200.0 or 25-30 gm.), chlorate of potash (50.0-60.0 or 5-10 gm.), resorcin (15.0-20.0 or 5-8.0 gm.), hydrochloric acid (one tablespoonful to a quart of water) or lime water (1-2 or  $\frac{1}{4}$  or  $\frac{1}{2}$  quart). Tar preparations must be avoided because they impart a disagreeable taste to the meat and would make it unpalatable in case slaughtering of the animal becomes necessary. The medicines mentioned above are to be given in moderate dilutions; however, in bloating of a high degree their administration meets with considerable difficulty, because the high pressure present in the rumen prevents deglutition and the restlessness of the patients causes danger of aspiration. The medicines may also be introduced through the esophageal sound or, after puncture of the rumen, through the tube of the trochar.

Lungwitz found in a comparative investigation of so-called absorbents 10% solution of burned magnesia most effective, next came lime water, spirits of ammonia (2%) and soapwater (2%); the last two also cause belching.

After the subsidence of acute symptoms, the animals should be subjected for several days to a strict diet, so that all easily fermenting material is removed from the rumen.



Moderate doses of salts or medicines stimulating the removal of the contents of the rumen are indicated. In secondary bloating, particularly if due to obliteration of the esophagus, puncture of the rumen cannot be avoided, in case the esophageal obstruction cannot be removed speedily.

**Prophylaxis.** Primary acute bloating is always due to careless pasturing or improper feeding and can be prevented with care and attention. It is necessary to educate shepherds and attendants by proper instruction. Since leguminosæ are most dangerous, it is proper not to pasture ruminants on such pastures, and the restraint should include barley stubble fields. Animals should be permitted to run on such fields only after they have received some dry feed or have fed on less luxuriant pastures, so that they will not take up too much of such juicy vegetable feed. They should never be left long in dangerous pastures and should be moved about during pasturing. The same precautions should be observed in feeding on very luxuriant pastures and particularly on dewy mornings or after rains.

Freshly-cut green feed is to be given in smaller rations and mixed with larger amounts of dry feed the younger and the more juicy such plants are. The same precautionary measures must be observed when feeding easily fermentable material (bulbous plants, floury feed, germinated malt, etc.). One should always avoid feeding material which is already fermenting or heated; if in this condition it should be cooled, dried and mixed abundantly with dry feed. The care-takers of animals should be made familiar with the simplest methods of expelling gas from the rumen. Friedberger & Fröhner recommend that farms and communities with large herds should always have trochars in stock.

**Literature.** Bouley, Dict., 1879, X, 156.—Eber, Z. f. Tm., 1906, X, 321.—Imminger, W. f. Tk., 1906, 221.—Leyendecker, B. Mt., 1890, 163.—Lungwitz, A. f. Tk., 1892, XVIII, 70.—Noack, S. B., 1896, 141.—Schlampp, Ther. Technik, 1907, II, 264.—Wöhner, W. f. Tk., 1905, 825.

### (c) Chronic Bloating of Ruminants. *Tympanitis chronica ruminantium.*

Chronic bloating of ruminants is only a group of symptoms which may be present in a variety of diseases of the fore-stomachs, the abomasum, the esophagus and the intestines.

(If chronic bloating is considered in a special chapter in spite of its purely secondary nature, this may be justified by the fact that the nature of the primary disease cannot always be ascertained in the living animal and the affection may require surgical interference.)

**Etiology.** Chronic tympanitis is observed in diseases in which the removal of gases from the rumen, either by belch-

ing or by the intestinal tract, is prevented for a considerable time. The amount of gas formed may be normal or the gas formation may be more lively than normal, without, however, reaching a high degree. Chronic bloating occurs preferably in atony of the fore-stomachs, both primary and secondary.

Another frequent cause is traumatic gastritis, while in calves and occasionally in lambs also, bloating is due to the occlusion of the openings of the fore-stomachs or of the pylorus by hair or feed balls. Disease of the abomasum likewise occasionally produces chronic tympanitis.

Another cause is stenosis of the esophagus (see page 231) which prevents the removal of rumen gases by belching, and also rumination. Stenosis of the esophagus is most commonly produced by tuberculous mediastinal lymph glands as pointed out by Johne. Weber and Goldmann have each reported a case of compression of the lower end of the esophagus by large echinococcus cysts of the liver. In a case of Ade, the lower end of the esophagus was compressed by the reticulum which had entered the thoracic cavity through a slit in the esophagus. Esophagus dilatation may also be the cause of chronic tympanitis, because the former leads to occasional temporary obstruction of the esophagus.

Hamoir is inclined to believe that it is not the stenosis of the esophagus itself, but the compression of the pneumogastric nerve fibers which supply the fore-stomachs that is the cause of chronic bloating (see also under stenosis of the esophagus).

Stenosis of the intestines may likewise be the cause of chronic tympanitis.

**Anatomical Changes.** Aside from the fundamental affection there is intensely fermenting food in the rumen, as well as large amounts of gases. The gases contain less carbon-dioxide than in acute bloating. In a case investigated by Erdmann, he found 42% CH<sub>4</sub>, 32% CO<sub>2</sub>, 20% N. and traces of hydrogen. Lungwitz found the proportion of hydrocarbons to carbon-dioxide as 1.1:1.2-1.5.

**Symptoms.** Chronic tympanitis consists in a gradually increasing, generally moderate, bloating of the left flank depression; it occurs at first at irregular intervals, later, however, and in profound cases, regularly after each feeding. It may exceptionally be present as a permanent condition. The degree of bloating is influenced by the character of the food; if the latter is very fermentable, bloating is more noticeable and may then be very extensive. The movements of the rumen may be perfectly normal or, in severe cases, they are somewhat diminished or decidedly sluggish. Rumination may be more or less suppressed or completely abolished. In the latter case other gastro-intestinal disturbances become manifest.



Eber wants the term chronic tympanitis reserved for those morbid conditions where the function of the stomach is not disturbed at all or shows a very minor degree of disturbance in spite of bloating of a high degree.

**Course.** Chronic tympanitis usually develops slowly and extends over weeks and months. Its course is determined by the underlying primary affection.

**Diagnosis.** The periodic appearance of generally moderate bloating is sufficient for a diagnosis. Since chronic tympanitis is only a symptom it is always desirable to diagnosticate the underlying primary affection. It is then important to remember that chronic tympanitis due to an affection of the esophagus or depending upon tuberculous mediastinal glands is characterized by periodically recurring attacks of bloating of the rumen without disturbance of the gastric function or in very severe cases with little disturbance; also that all symptoms cease after the removal of the gases through the esophageal tube or through puncture of the rumen (see page 233). In chronic bloating due to intestinal stenosis the gastric function is disturbed and disturbances of defecation are particularly well marked.

When other gastric disturbances are associated with chronic tympanitis, primary or secondary disease of the fore-stomachs must be looked for (atony of the fore-stomachs, foreign bodies) or of the rennet.

**Prognosis.** Chronic tympanitis does not endanger life *per se*; however, its periodic appearance disturbs the nutrition to a certain degree, but the emaciation which finally comes on is really due to the underlying primary disease. The latter also determines the prognosis.

**Treatment.** The removal of the fermenting food may be accomplished by stimulation of the movements of the rumen through massage of the rumen in an elevated position and by the administration of certain medicines, mentioned before (see pages 253 and 254). In addition laxative salts may be given (200.0-300.0 gm. or 10-25.0 gm. for smaller animals) combined with tartar emetic (2.0-3.0 gm. or 0.3-0.5 gm.), aloes (5-10.0 gm. or 2-3.0 gm.) or ipecacuanha (4-6.0 gm.). Such medicines must be given repeatedly. Anti-fermentative solutions (see page 261) are also useful, particularly disinfectants, and eventually also lavage of the rumen (see page 274). After the morbid condition has been removed, one should administer, with proper feeding, some more laxative salts, but in smaller doses (30-50.0 gm. or 5.0-10.0 gm.). Good effects are also seen from chloride of barium (6-8.0 gm.) dissolved in water (Faber, Marder).

In the presence of an incurable underlying condition, it is advisable to slaughter the animals. If there is reason for suspecting a foreign body in the stomach, incision into the rumen



may bring about recovery; this procedure should particularly be considered in the periodic tympanitis of calves.

**Literature.** Ade, M. t. W., 1909, 62.—Albrecht, W. f. Tk., 1890, 149.—Eber, Z. f. Tm., 1906, X, 321.—Erdmann, A. f. Tk., 1875, 1, 289.—Faber, D., 1908, 330.—Imminger, W. f. Tk., 1906, 4.—Johne, S. B., 1881, 24; 1886, 56.—Lungwitz, A. f. Tk., 1893, XIX, 75.—Peschel, S. B., 1884, 98.—Weber, D. t. W., 1909, 347 (Lit.). (See also literature on the primary underlying diseases.)

**(d) Atony of the Fore-stomachs. Atonia ruminis, reticuli et omasi.**

(*Chronische Unverdaulichkeit, Chronische Indigestion, Chronische Dyspepsie, Chronischer Magendarmkatarrh des Rindes* [FRIEDBERGER & FRÖHNER]; *Pansenlähmung* [DIECKERHOFF]; *Ermüdung* [Parese] *des Verdauungskanal* [Pütz]; *Löserverstopfung, Sporadische Löserdürre, Chronische Entzündung des Löser* [ANACKER] [German]; *Indigestion chronique des ruminants, Obstruction ou Engouement ou Indigestion du feuillet, Omasite ou Gastrite chronique, Météorisation chronique* [French].)

Atony of the fore-stomachs consists in a slowly developing weakness of contractions of these organs, depending upon a diminution of irritability and energy of contraction. The removal of the normal or even subnormal amount of food becomes more or less retarded and digestion disturbed.

**Occurrence.** The disease is most commonly observed in cattle, somewhat less frequently in goats, occasionally in sheep. The frequency of its occurrence depends upon the prevailing conditions of feeding. The disease is frequently met among animals of poor people, in the neighborhood of larger cities, or in such parts where milch cows and cattle in general are fed permanently with various substitutes for natural feed or with relaxing or, on the contrary, with irritating feed. Most cases occur during the colder season, especially in spring, because then an irrational method of feeding is mostly resorted to. During certain seasons or in districts with poor crops the disease is particularly common. Finally, the disease is seen in some parts in very young calves if, after being fed with milk, they are given substitutes or other improper food.

**Etiology.** As a **primary** acute affection we find atony of the muscularis of the first three segments of the stomach after a sudden change of food (after change of owner), as a consequence of overexertion during long transit, or after difficult parturition. To this class also belongs the affection described by Janssen as chronic gastric catarrh of cows after parturition, which attacks particularly good milch cows. An insufficient

function of the muscularis of the fore-stomachs also occurs after intense psychic stimuli (excitement, pain, longing for the calf). Atony due to these causes is probably combined with lack of secretion in the abomasum and usually disappears in a short time, but if neglected it may lead to the more serious chronic atonic condition. The same may be said of the acute atony seen after the course of acute, febrile affections or after parturient paresis which may last for some time and which may be intensified by the aspiration of the contents of the reticulum.

In its usual chronic form atony of the fore-stomachs may occasionally be caused by dilatation of the rumen, particularly if this affection takes a protracted course, in consequence of which the muscle fibers become paretic.

Usually, however, the affection is due to prolonged improper feeding. As harmful in this respect must be mentioned food which is dry, not easily digestible and not very nutritious, such as chaff, straw, marsh hay, garbage from the garden or the kitchen, particularly if it contains fragments of bones, also oil cakes, bran, shelled grain or leguminosæ. Not less dangerous are tough, fibrous food stuffs, such as coarse alfalfa, raspberry bushes, brush hay, because such materials are liable to accumulate in the rumen. The obnoxious effect of such feed upon the function of the stomach may be increased by a deficiency in the ingestion of water. The disease is liable, on the other hand, to make its appearance if the animals have been fed for a long time with fermentive, spoiled, wet or frozen feed; also with boiled potatoes or with a thin floury mash (Eberhardt) without any rough feed at all.

Other causes that may be enumerated are insufficient mastication and mixing of the food with saliva, in consequence of faulty teeth or in chronic disease of the tongue, greedy feeding, or the frequent prevention of rumination in work cattle.

The affection develops in young calves in connection with gastro-intestinal catarrh, if these animals, after having previously been fed exclusively with milk, receive too suddenly or too early, bread, crushed oats, germinating malt, hay, etc., in addition to skimmed milk (Imminger).

Predisposing causes are early youth and old age, and debility after chronic, acute and febrile diseases.

**Secondary** atony of the fore-stomachs cannot usually be differentiated clinically from the primary form. An exception is that form of secondary atony which is observed in the course of traumatic gastritis, and other forms of inflammation of the fore-stomachs or of the abomasum. For this reason and in consideration of etiologic factors, it will be treated in a special chapter (q. v.).

In the advanced stages of pregnancy all sections of the stomach are sometimes so compressed by the pregnant uterus that atony results. More extensive adhesions of the fore-stomachs among each other, or with neighboring organs, diminish

the functional activity of the fore-stomachs, as does also prolapse of the omasum into the thoracic cavity. Other causes are tumors (see tumors of the stomach), diffuse lymphadenitis of the wall of the fore-stomachs or of the abomasum (Eber), certain diseases of the abomasum (obstruction by foreign bodies or feed material, tumors, ulcerations), subacute or chronic peritonitis, even if it has not caused adhesions of the fore-stomachs, chronic disease of the liver (chronic interstitial inflammation, echinococcus, tumors, gallstones), certain chronic diseases of the lungs and heart.

**Pathogenesis.** In consequence of the enumerated causal conditions, especially, however, in consequence of continued improper feeding, there occurs a diminution of the irritability or of the energy of contraction of the fore-stomachs, or both functions of the latter become diminished. The resulting weaker and less frequent contractions of the fore-stomachs are the cause of a deficient mixing and of a retarded removal of the contents. According to prevailing conditions and amounts of the feed contents, the degree of disturbance of function and the amount of ingested water, there occurs either a lively formation of gas or the contents desiccate more or less; even in the latter case formation of gas is rarely absent. Every additional act of ingestion of improper food further increases the disturbances.

No matter in what section the primary disturbance, desiccation of the feed always occurs in the rumen and reticulum, that is, in those sections where it is even normally less rich in water than in the omasum where the contents rarely desiccate.

The reason for this behavior lies in the functional inter-relation of the individual sections of the stomach; stoppage of movements in one section also causes stoppage of movements in the other sections. It can not be denied, however, that occasionally the reticulum alone is the seat of the obstruction. This occurrence is plausible since normally the contents of the reticulum are comparatively firm, its muscularis weak, and since finely divided feed (short cut chaff, bran, etc.) may occasionally get directly into the third section of the stomach without rumination. These are however rare cases and their differentiation from other forms of atony appears impossible since disease of the reticulum at once also leads to disturbance of the functions of the other sections of the stomach.

Incomplete mixing and accumulation of feed masses lead to disturbances of digestion and to lack of nutrition, and these again further increase the atony. Abnormal fermentation of feed in the fore-stomachs may form poisonous decomposition products, while the continued or often repeated dilatation leads to a permanent dilatation of the fore-stomachs. If the affection has lasted a long time, there also result inflammatory or even necrotic changes of these sections. For these reasons catarrh of the abomasum and of the small intestines is one of the common sequelæ and the deeper inflammatory processes may spread from the walls of the fore-stomachs into the peritoneum.



**Anatomical Changes.** Rumen and reticulum occasionally also the omasum, are more or less dilated. The increase in size of the reticulum is particularly obvious, although in young calves, the rumen is most enormously dilated (Imminger). The contents of the reticulum are unusually dry, almost rocklike, but they can be ground up into a fine powder between the fingers; they completely fill the reticulum ("*Löserverstopfung*" [German]). The rumen likewise usually contains desiccated, fetid masses of feed and usually also gases. If the affection has been caused by feeding thin mashed, or boiled potatoes, the contents of the rumen look like street mud or like a thin dough (Sipp, Eberhardt), while the reticulum may not be obstructed, as is occasionally the case in other cases of atony.

Desiccation of the feed-mash in the reticulum without dilatation of the organ is a common but negligible finding in severe febrile general diseases or other affections in which the movements of the fore-stomachs have been diminished for a considerable time.

The mucosa of the fore-stomachs either does not show any changes at all, or it appears reddened uniformly or in spots, occasionally with hemorrhagic spots, if the process has lasted for some time. These changes can easily be seen postmortem, since the cornified squamous epithelia soon after death become detached in shreds or adhere to the masses of feed (common postmortem appearance!). One occasionally finds pressure-necrosis or hemorrhagic ulcers in the reticulum, usually combined with a general acute peritonitis. The latter, however, may be present without pressure-necrosis of the wall of the reticulum.

In secondary atony the causative underlying disease can generally be ascertained on postmortem examination.

**Symptoms.** In the **acute form** of the disease one only notices a diminution of the appetite and thirst; rumination is infrequent and sluggish and there is a decrease in the energy of the rumen movements which are, however, normal in frequency (simple indigestion [Eber]). If these symptoms, which are not very prominent, are overlooked, and if the animals are not properly treated, the clinical picture of the chronic form gradually develops.

The **chronic form** usually develops very gradually, although it may occasionally evolve within a very few days into a characteristic, clinical picture. The animals from time to time take less feed and they generally prefer rough feed; in some cases the appetite may be perfectly normal for several hours and even for a few days. Thirst is generally diminished, but it may also be increased (Bouley, Heu). Here and there the animals lick greedily. Rumination is, as a rule, gradually diminished and it may also occasionally become suppressed suddenly. As a rule rumination only occurs from time to time; after a longer inter-

val the masticatory movements are sluggish and reduced in number. Belching occurs rarely and it expels fetid gases, becoming more frequent after more pronounced meteorism. Gagging or vomiting is rare; if the latter does occur, it expels a thin-mashy, fetid material.

The circumference of the abdomen is frequently increased on the left side and below, with bloating of the left flank, which may even become quite prominent; if, however, there is a continuous lack of appetite, the circumference of the abdomen may be diminished. The rumen is usually larger, even in deficient digestion of food, sometimes only moderately filled or almost empty; it generally feels hard, rarely fluctuating. The upper part of the rumen is filled with gases, so that the consistency of the solid contents of the rumen can only be ascertained by strong pressure upon the left flank or by rectal palpation, or in smaller animals by simultaneous palpation on both sides. Bloating of the rumen usually remains within moderate limits; it is, however, subject to considerable variations because it increases more or less after the ingestion of feed, diminishing again or even entirely disappearing (chronic meteorism). Every improvement of the appetite with an increased ingestion of feed promptly leads to an increase of meteorism which may become permanent if the gastric movements cease entirely. The movements of the rumen at first are diminished considerably in frequency and energy, so that the disturbance may easily be overlooked; without proper treatment the movements of the rumen may cease entirely after a few days. Disturbances of the movements of the rumen are subject to similar variations as meteorism; pressure upon the rumen sometimes elicits pain.

An increased resistance may, according to Detroye, be felt in the abdominal cavity in obstruction of the reticulum if the fist suddenly makes firm pressure behind the right costal arch; the animals simultaneously emit a dull groan. Hink states that the reticulum is tender to pressure when percussion is made over the lower posterior portion of the thorax, or to pressure upon the ensiform cartilage.

The rumen and intestinal sounds are suppressed, the latter, however, are loud and rumbling before the appearance of diarrhea. Defecation is less frequent and is finally obstinately suppressed. The feces are peatlike, blackish, their surface shining and they contain poorly digested food particles. Exceptionally, that is if the disease has been caused by feeding of mashy substances, no disturbances of defecation may be noticeable. Sooner or later constipation yields to malodorous diarrhea which may be permanent or may alternate with constipation. Sometimes mild colics supervene, particularly before diarrhea sets in.

The temperature remains below 40° C. as long as there are no complications. The pulse is at first normal as to frequency and strength, but becomes rapid and weak later on. The respiration is more or less accelerated, particularly if bloating exists,

and expiration is accompanied by groaning and sighing, particularly on lying down. There also occurs not uncommonly grating of the teeth. In one case Petersen observed a dry cough after the disease had existed eight days.

The sensorium frequently does not manifest any disturbance for a long time, even if emaciation is already present, except perhaps that the animals are rather sluggish and lie down a good deal. Sometimes the patients are somewhat excited and show twitching (Petersen).

In the further course of the disease or with complete lack of appetite, the general debility becomes more and more marked and the gait becomes staggering. Exceptionally one observes in male animals, also in cows, parietic conditions similar to the prostration of cows (Wucher) or to parturient paresis (Sticker, Schell, Eber).

In the further course, emaciation and weakness become more and more manifest, the eyes sink in, the hair becomes harsh, the skin dry and the dental pad, which is dry from the start, becomes furrowed with clefts. The secretion of milk decreases and finally stops entirely.

In secondary atony we are sometimes able to recognize symptoms due to the underlying fundamental condition.

Chronic gastro-intestinal catarrh of cows after calving begins within the first week after parturition with sluggishness of the gastric movements and diminution of appetite; soon marked emaciation, which is out of all proportion to the diminution of appetite, becomes noticeable. A peculiar sweetish smell emanates from the mouth and pervades the whole barn. The milk likewise has a peculiar disagreeable smell. The disease lasts several weeks and if not treated properly leads to cachexia and death. Inflation of air into the udder leads to speedy recovery which may also be caused though not in all cases by the administration of certain medicines.

In **young calves** the disturbances appear a few days after improper feeding (Imminger). The first symptom is violent diarrhea which may last for eight to ten days and may then stop suddenly. The fluid feces then become firm and gray and have an intensely fetid odor. As soon as scouring stops, the general condition is much disturbed and the previously good appetite is suppressed. Then chronic meteorism appears either very gradually or on the contrary very rapidly, and it is so intense that the rumen becomes much dilated and presses upon the rectum (Bitard). As a rule bloating occurs after each feeding and lasts six to twenty-four hours. As bloating occurs the animals become listless and curve their back. An elevation of temperature also occurs. After some time considerable emaciation is observed.

**Course.** Aside from acute cases which under proper treatment and diet end in recovery almost without exception, atony which has become chronic has a very variable course, and temporary improvements alternate with aggravation at variable intervals.



Permanent improvement may be looked for if simultaneously with the appearance of flat, grumous masses of feces, supposedly coming from the reticulum, the bloating disappears, the rumen does no longer contain firm masses, its circumference becomes diminished, its motions become more frequent and more energetic, and the groaning and sighing subside. In adult animals improvement is very gradual and leads to complete recovery only after four to twenty-five days (Cadéac); however, it may be interrupted by a recurrence by improper attendance on the part of the owner of the animal. Atony due to compression by the uterus disappears within a few days after calving, in favorable cases. If properly treated, calves likewise recover within a few days (Immingier).

Unfavorable signs are entire absence of appetite and of the motions of the rumen, continuous bloating, constipation resisting every method of treatment or, on the contrary, continued diarrhea, fever or subnormal temperature, marked tenderness on pressure of the rumen, continuous sighing, inability to rise, a paretic condition, convulsions, cachexia.

The duration of the disease is from several weeks to several months (after Bouley twenty-five to thirty days, after Cadéac one to two months, after Dieckerhoff usually many months, even one to two years). Sometimes, however, the affection only lasts eight to ten days (seen by Eber in cows in advanced pregnancy).

**Diagnosis.** The clinical symptoms and the frequently helpful history of the case usually permit a diagnosis of atony of the fore-stomachs. Severe general diseases which in their course also lead to a diminution of the movements of the fore-stomachs, are from the start distinguished by fever and other prominent symptoms and also by their rapid onset. Dilatation of the rumen occurs after excessive feeding of animals which were previously healthy. The rumen is considerably dilated; if the condition is at all severe, its movement cannot be seen at all. Acute bloating could only be confounded with those cases of atony of the stomach where there has accidentally been a higher degree of meteorism. The rapid development of bloating following the ingestion of easily fermenting food in large amounts by a previously healthy animal, and the complete recovery in the shortest time after proper treatment, easily secure the diagnosis of acute meteorism. The forms of chronic bloating not depending upon disease of the fore-stomachs or abomasum (see page 263) are characterized by the fact that a collection of gas is the most prominent or even the only symptom, while the function of the fore-stomachs suffers only during strong bloating; besides all symptoms disappear for the time being as soon as the meteorism has been relieved. Acute gastro-intestinal catarrh and gastro-intestinal inflammation can be differentiated by their usually sudden onset, by fever, by the

depression in the left flank, and the absence of gas and masses of feed in the rumen; in gastro-intestinal inflammation, there are from the start signs of grave general disturbance present. If, however, acute gastro-intestinal catarrh or gastro-intestinal inflammation have developed in consequence of atony of the fore-stomachs, then the history only can clear up the case.

It is usually easy to decide whether primary or secondary atony of the fore-stomachs is present if one considers the history of the case, the condition of the other organs, and also the manner in which the affection is influenced by treatment. Gastric atony may be assumed to be due to compression by the pregnant uterus, when towards the end of gestation disturbance of digestion develops gradually without any errors of diet and when the uterus is unusually large. Prolapse of the omasum into the thorax produces a dull or tympanitic sound upon percussion of the lower third of the thorax on one or on both sides, while in place of the absent respiratory sound, strong peristaltic movements may be heard (Harms); there are also occasionally circulatory disturbances such as dilation of the jugular vein, diminution of the apex sound on one side (Liénaux); these are due to compression of the heart. In cases of secondary gastric atony one may observe the symptoms of chronic disease of the liver, lungs or heart or those of subacute or chronic peritonitis. However, frequently the seat of the primary affection cannot be recognized in the living animal.

The early recognition of traumatic gastritis which is indeed only a special form of secondary gastric atony is very important. Aside from the tenderness to pressure of the region of the omasum which is frequently present, and the sudden onset of obstinate gastric disturbances without any apparent cause, which will resist any and every treatment, the appearance of spontaneous pains after drugs which stimulate gastric movements (see page 282) point to a traumatic gastritis. Sometimes only the further course brings enlightenment, especially the subsequent affection of the pericardium, the pleuræ, the lungs and the subcutaneous connective tissue in the sternal region.

**Prognosis.** This depends primarily upon whether a primary or a secondary atony exists, upon the duration of the disease and upon the general nutrition of the animal. Primary gastric atony can generally be cured by timely treatment and proper regulation of diet. The prognosis of secondary atony depends upon whether the underlying primary disease is curable or not. Atony due to gestation usually disappears a few days after parturition, but even in this form death may occasionally occur (Eber, Frasch).

Aside from the last mentioned form of gastric atony, recovery cannot be looked for after the disease has lasted for

two to three weeks and if the gastric disturbances do not yield to prolonged treatment, if constipation has existed several days or scouring several weeks, if a paretic condition has set in or if symptoms of gastro-intestinal inflammation or of peritonitis make their appearance. Then the animals should be slaughtered. In tuberculous cows or in animals advanced in gestation the prognosis ought to be guarded even in primary atony (Eber).

**Treatment.** It is of primary importance to regulate the diet according to the same principles as were laid down for overfilling of the rumen (see page 253). After withholding rough feed for one or more days, one feeds for a few days only good hay in small quantities and some floury fluids mixed with some common salt. Feeding until the animals are satisfied and at once after the return of the appetite always brings about a considerable relapse of the bad condition. Pasturing has a good dietetic effect. Calves do best on boiled milk or boiled oatmeal, which should be given in small quantities several times a day (Imminger).

The treatment proper consists preferably in massage of the rumen (see page 253). Drugs stimulating motion of the rumen (see page 254) are likewise very serviceable and are eventually useful for a long time. Other laxatives should only be given in complete constipation, because these drugs have no effect upon the motions of the rumen, and if administered for some time in large quantities of water, they are liable to increase the disturbance of the stomach. Diarrhea of somewhat longer standing ought to be combated by the administration of styptics.

A drug which has proved beneficial is hydrochloric acid (one to two tablespoonfuls in a quart of water several times daily); it does not only stimulate the digestive processes, but also hinders to a certain degree the abnormal decomposition in the stomach. This is also accomplished by creolin (5 gm. three times daily in a bottle of water) which has been recommended by several observers. The following drugs are also to be mentioned in this connection: Common salt and sodium bicarbonate  $\bar{a}\bar{a}$ ; sulphate of sodium; artificial Carlsbad salt (one to two tablespoonfuls).

Cases which have not done well under any other mode of treatment have sometimes been benefited by the administration of large amounts of water directly into the rumen through a trochar introduced through the left flank and connected with a rubber tube and a funnel. In this manner it is possible to introduce up to fifty quarts of water into the rumen of cattle within the course of several hours (Deffke); it may be well to add 8 to 10.0 gm. of hydrochloric acid or 8 to 15.0 gm. of creolin, lysol, resorcin. However, if there is any obstruction of the reticulum or an obstruction between the sections of the



stomach the introduction of such quantities of water acts unfavorably (Engesser). In obstinate cases of obstruction of the rumen or reticulum there remains as a means of last resort the incision of the rumen (see page 255); after it has been made, it is possible to clean out the rumen and then to irrigate the obstructed reticulum.

Imminger was able to get permanently good results in young calves by the irrigation of the rumen followed by the proper regulation of the diet.

**Irrigation of the rumen** in calves is, according to Imminger, performed as follows: A trochar 10 cm. long with a canula 8 mm. in diameter is provided with a rubber tube 1 meter long, carrying on its free end a funnel. The puncture is made with the trochar which is then withdrawn and the rubber tube is attached to the canula. A 3% warm common salt solution (30 to 35° C.) is allowed to run into the rumen until the fluid remains in the funnel. The solution is left in the rumen for some time, then the rubber is detached from the canula, but the latter is left in place in the rumen. Next the calf, whose hind and front legs have been tied together in pairs, is lifted by two or three persons upon its left side on two tables, placed side by side, so that there is left an opening of a few inches between them which will permit the canula to be in the free space. The fluid will at once begin to be voided and this may be assisted by mild pressure upon the upper side of the calf. Finally the canula is slowly withdrawn, so that all fluid which had been introduced can run out. After the removal of the dirty brown, stinking irrigation water, the rumen is filled a second time, but the fluid is now not withdrawn, but is, by proper massage, pressed into the other two sections of the fore-stomachs, the abomasum and the small intestines. After the contents of the rumen have been diminished in this manner, the canula is removed. The next day there is moderate diarrhea with fetid defecation and then recovery takes place.

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(e) **Foreign Bodies in the Fore-stomachs. Corpora aliena**  
reticuli, ruminis et omasi.

(*Traumatische Hauben-, Haubenzwerchfellentzündung, Traumatische Indigestion, Traumatische Pansenlähmung*  
[German] [EBER].)

**Occurrence.** Foreign bodies and diseases caused by them are met with especially frequently in cattle. Particularly cattle having female attendants, and cattle of poor owners are affected, as these animals have usually the best chance of taking up foreign bodies. Still more frequent are cases in the barns of tradesmen and factories where work is done with nails and

needles (shoemakers, needle makers, iron workers, spinning establishments). Sometimes gastric disturbances due to foreign bodies appear in a herd in an almost enzootic manner.

According to Eber's statistics of 235 correctly reported cases of paresis of the rumen (overfilling of the rumen, primary and secondary gastric atony) in cattle, 17.9% were due to foreign bodies.

Among other ruminants (goats, sheep, buffaloes) foreign bodies are more rare, being comparatively most common in goats.

**Etiology.** The frequent occurrence of foreign bodies in the fore-stomachs of cattle depends upon the habits of these animals to lick all possible objects accessible to them and often to swallow them. Besides various objects are found in the food containers, particularly in the poorly kept barns of small farmers, which have gotten there with the garbage, the sweepings, etc., or which are derived from the wardrobe of female attendants. Such objects are then easily swallowed with the feed, the more so since cattle chew their food only very superficially and coarsely before swallowing. (There is a case on record of a perforated but otherwise complete egg shell being found in the rumen of a cow.) Besides, the long papillæ of the tongue projecting posteriorly prevent foreign bodies from falling out of the mouth. But even with good care the feed may contain foreign bodies such as pieces of wire in pressed hay, nails or pieces of wire which may have been derived from burned wood and may have been swallowed in the pasture.

Smaller foreign bodies vary a good deal as to their nature and form; most dangerous are those of iron because they are not changed by the juices of the fore-stomachs. Needles, pieces of wire, nails, blades of knives, pieces of scissors are found most commonly; less common and less dangerous are splinters of wood, larger pieces of wood, leather, cloth, balls, pieces of money and roots.

Licking of the wall of the barn, pasturing on marshy places with the ingestion of marshy or dirty hay often brings sand in large quantities into the fore-stomachs and sometimes causes extensive disease of cattle, particularly in inundated territories (observed by Marconi in buffalo calves and by Müller and Prietsch in cattle). Drinking in shallow bodies of water may also cause overfilling of the fore-stomachs with sand.

Hairballs, which are to be looked upon as foreign bodies, occur not uncommonly in the rumen, the omasum, occasionally also in the abomasum of cattle and of other ruminants. Hairs or woolen fibers which are taken up while licking their own body or the bodies of other animals unite with vegetable fibers to form spherical or ovoid bodies (Bezoare, agagropili) and become covered by a brownish, smooth crust formed by mucus and salts. Hairballs sometimes attain the size of a fist and



are most frequently encountered in the stomachs of adult cattle, sometimes also in calves and lambs. Eating of wool particularly, as well as itching diseases of the skin, give rise to the formation of hairballs. In lambs fed with cakes of unshelled cotton seed or with very woody beets, feedballs are formed by the undigested residues which are similar to hairballs (Moussu). Foodballs are commonly found in the abomasum.

In the stomach of a still-born calf Schell found several hairballs up to the size of a chestnut; Mégnin found hairballs the size of a fist in an eighteen-day-old calf.

**Pathogenesis.** **Blunt foreign bodies** only rarely lead to bad consequences for the animals; but these occur when the foreign bodies are pressed into the communications between the sections of the stomach and intestines or into the pylorus; or when they are as large as a nut, so that they prevent free communication between the fore-stomachs or between abomasum and intestines, or if they prevent the motions of the fore-stomachs. In all these cases the removal of the feed-mash and of the gases suffers more or less and the muscles of the fore-stomachs become sluggish (see page 265). Pressure exerted upon the wall of the stomachs by heavy foreign bodies may bring about nutritive disturbances on the parts of the affected wall, and finally inflammation which may spread to the peritoneum.

The effect of **sharp** or **pointed bodies** depends upon their nature, form and length and also upon their location in the stomachs. Larger foreign bodies are, in general, less dangerous than smaller ones, because they do not so easily get from the rumen into the reticulum and may remain for a long time embedded in the feed-mash in the rumen without producing any evil effect. (Bergmann found a kitchen knife in the rumen which had been there three months without causing any damage.)

Foreign bodies, as a rule, become wedged into the wall of the comparatively small but vigorously contracting omasum, while the rumen, the reticulum and the abomasum are rarely injured. Pointed bodies of inconsiderable length (nails, pieces of wire, hairpins), particularly if provided with a head or bent over, get more or less deeply into the wall of the stomach, but they do not penetrate further. However, long and pointed bodies, smooth along their whole extent (such as long nails or pieces of wire, darning, embroidery, suturing or tobacco needles) will be transported not only to neighboring, but even to distant organs by the contractions of the stomachs and the diaphragm and by the abdominal pressure (especially during parturition and during restraint before an operation) or by pressure of the uterus if this is well along in gestation. Since perforation usually occurs in that part of the wall of the omasum which is directed towards the diaphragm, these foreign bodies usually penetrate in the direction of the diaphragm or heart.



Perforation and continuous irritation of the wall of the stomach first causes pain, which, however, is fairly acute only if the penetration took place more or less rapidly. The injury of the wall of the stomach opens the way to the bacteria which are contained on the foreign body and in the gastric contents, and these microbes then cause inflammation in the neighborhood of the penetrating wound. Circumscribed inflammation usually spreads to the serous covering of the injured section of the stomach, even in those cases where the penetrating foreign body remained lodged in the wall of the stomach. If the foreign body penetrated rapidly, the inflammation has an acute character, otherwise it is chronic, but it may also spread to the whole of the peritoneum. A perforating foreign body may exceptionally injure a larger blood vessel.

The motions of the fore-stomachs are interfered with both by the injury to the gastric wall itself, and also by the acute inflammatory process, and later on mechanically by adhesions which are formed (Eber's traumatic paresis of the rumen).

**Anatomical Changes.** **Blunt foreign bodies** are, as a rule, found either imbedded in the feed-mash in the rumen or they are wedged into the openings of the stomachs or the pylorus. Sand or gravel may become deposited on the internal surface of the rumen, more rarely upon the other sections of the fore-stomachs. (Wilhelm found 120 pounds of sand in the fore-stomachs, the walls of which had become perfectly hard, so-called "*Magenversandung*" [German] Saburra.) Occasionally one sees inflammatory changes of the mucosa of the fore-stomachs, exceptionally a circumscribed peritonitis (Roeder, Wilhelm).

**Pointed foreign bodies** cause changes preferably in the wall of the reticulum (traumatic inflammation of the reticulum, reticulitis traumatica). On post-mortem examination loss of substance, with hyperemia in the neighborhood, small hemorrhages, also purulent inflammation are found, sometimes in the mucosa only, at other times in the deeper tissues of the gastric wall, or there may be a cicatrix only or a fistulous tract surrounded by cicatricial tissue. In other cases the foreign body has penetrated further into the wall of the stomach or has perforated it, then a circumscribed fibrinous peritonitis is found in the immediate neighborhood of the injury, and an agglutination or adhesion of the reticulum to the diaphragm (traumatic diaphragm-reticulum inflammation) or to the rumen. Frequently one will find a fistulous tract in the cicatricial adhesions which communicates with the reticulum, or the cicatricial tissue includes tough-walled abscesses containing usually ill-smelling pus which is occasionally mixed with food particles. The fistulous tract usually contains the rusty or dark discolored foreign body, but this may also have become disintegrated.

The pointed, long and smooth foreign bodies frequently penetrate from their first place of perforation into other organs, most frequently towards the heart, more rarely towards the lungs, liver, spleen, muscles of the thigh; there are then formed cicatricial bands leading to the reticulum, which may be interrupted by abscesses. In those very rare cases where a foreign body has made its way to the outside world, there is formed a thick-walled fistulous tract leading from the region of the ensiform cartilage to the reticulum.

Foreign bodies are rarely found in the omasum or abomasum; they get there from the reticulum and sometimes leave the body through the intestines. Exceptionally a foreign body may travel over another route and penetrate through the left hypochondriac region, through the flank, or through the muscles of the hind leg; the road traveled is then also indicated by cicatricial tissue or by a fistulous tract.

Small hemorrhages at the site of the injury are common and the grayish discoloration in the newly formed connective tissue is due to these extravasations. Exceptionally internal hemorrhage may be noticed (Eggeling, Bräuer, Harms, Holterbach), then the reticulum and the next sections of the stomach, also the intestines contain blood coagula. Sometimes we encounter purulent ichorous peritonitis or pleuritis in consequence of the breaking of an abscess in the neighborhood of the reticulum. General purulent or sero-fibrinous peritonitis may also be developed if the gastric wall has been perforated without the prior formation of sufficient preliminary agglutination or adhesion; in such cases bacteria enter the peritoneal cavity from the reticulum. Sometimes we find on post-mortem metastatic foci in the internal organs.

**Symptoms.** Blunt foreign bodies sometimes produce an atony of the fore-stomachs (chronic dyspepsia) which cannot be distinguished from other forms of gastric atony and which therefore can only be diagnosticated if the history of the case furnishes some data. After the ingestion of sand (so-called "*Magenversandung*") or gravel, there are also present symptoms of chronic gastric atony (see page 268), and in some cases the wall of the rumen feels as hard as a rock; the sick animals constantly make empty masticatory movements, saliva dribbles from the mouth, the back is curved and a painful groaning is frequently heard (Krichels). The feces sometimes contain sand. In cases of sand in the rumen Wucher saw a paresis similar to parturient paresis of cows. In severe cases the disease leads to exhaustion and sometimes ends fatally after two to three weeks, but it often lasts for months.

The sudden stoppage of the openings between the fore-stomachs or the pylorus by foreign bodies, especially hair or food balls, occasionally occurs in calves and lambs and exceptionally in adult cattle. Such stoppage causes bloating which

appears suddenly and increases rapidly, great restlessness, spreading of the front legs, loud groaning, anxious expression, later on listlessness, almost complete inability to walk, increase of pulse and respiration, even eclamptiform attacks, sometimes vomitory movements which may cause the expulsion of the obstructing hair or food ball, and may in this way bring about a rapid recovery. Nevertheless, young animals usually succumb in one to two days (Richards saw three to five lambs die daily in a herd of 120).

The penetration of **pointed bodies** leads to inflammation of the stomachs (traumatic gastritis and inflammation of the diaphragm) and causes profound gastric disturbances, and in those cases which have not developed slowly, symptoms pointing to tenderness of the stomachs and of the diaphragm.

The time interval between the ingestion of the foreign body and the appearance of the gastric disturbances is very variable according to the nature of the foreign body. Two oxen experimentally received some medium sized nails pointed at both ends; they were introduced through the esophageal sound. One animal showed symptoms after twenty-four hours, the other only after four days (Marek). Koppitz inclines to the belief that at least four days elapse on an average between the ingestion of a foreign body and the appearance of the gastric disturbances, because the foreign body, which is usually introduced into the rumen, will pass into the reticulum only after this time. If the foreign body is accidentally taken up with mashy or fluid feed, it may get directly into the reticulum and penetrate into its wall.

The clinical picture after the ingestion of pointed foreign bodies is initiated by sudden disturbances of digestion without any apparent cause; in spite of preceding perfectly normal condition, the animals will cease to eat and to ruminate. At the same time pain becomes manifest. The animals betray much anxiety in their whole behavior, they do not like to move and issue plaintive sounds when lying down, getting up, turning rapidly, or in taking a step. Their gait is careful and stiff, particularly down hill on steep paths, because these conditions increase the pressure upon the diaphragm. Pain also increases on elevation of the hind legs (Imminger). In some cases colicky symptoms become manifest, such as kicking towards the abdomen, elevation of the feet, switching the tail, etc. Strong pressure upon the region of the ensiform cartilage excites pain which may also be present upon percussion and palpation along the insertion of the diaphragm, along the costal arches, but especially towards the ensiform cartilage.

The respiration is superficial and accelerated, the pulse rapid and tense. The temperature rises if the foreign body penetrates rapidly, otherwise it remains normal. The secretion of milk is suppressed.



The affection begins exceptionally with gagging or vomiting, with the expulsion of either blood or food mash through the mouth and nose (Müller, Wilhelm, Sequens, Holterbach). Gagging and vomiting are however repeated occasionally. The animals may die in consequence of the aspiration of feed-mash into the larynx. Exceptionally fluid or coagulated blood is passed per rectum. In a case of Harms the patient voided a red coagulum on the second day, which may clearly have been a cast of the reticulum. Rectal hemorrhage is, however, either transitory or may, like vomiting of blood, speedily lead to death (Eggeling, Holterbach). In a case of Eber in which the wall of the rumen at the entrance of the esophagus had been perforated by the foreign body, the cow fell sick suddenly under the picture of a paralysis of the tongue with inability to swallow. It occurs more frequently that traumatic gastritis is initiated by the clinical picture of overfilling of the rumen or of meteorism.

When perforation of the gastric wall has occurred slowly, pain is insignificant or absent. In such cases the disturbances of nutrition and the further course of the disease are in general identical with those of chronic gastric atony (chronic dyspepsia) (see page 268). Sometimes it is possible to demonstrate a circumscribed dullness before or behind the posterior boundary of the lung, which is caused by an abscess or by granulation tissue. The disturbances of digestion which vary from case to case have one feature in common, namely, a high degree of obstinacy. Still, temporary improvement or aggravation may occur (the latter particularly after deliveries and transportation). Emaciation and debility become very marked in consequence of digestive disturbances. The production of milk is usually considerably diminished from the start or even completely abolished.

In this protracted course of the disease fever often occurs later on. Schultz found an increase of leucocytes in the circulating blood and these findings in obstinate gastric disturbances speak in favor of a traumatic gastritis.

Complications arise frequently in consequence of the wandering of the foreign body, exceptionally also in animals which at first did not show any symptoms at all. Comparatively frequent is traumatic pericarditis, less frequent pneumonia or peritonitis. Sometimes the scene is closed by a general infection with septicemia or pyemia, sometimes again the bacteria are carried along and deposited in distant organs, for instance, the joints where they then produce local inflammatory processes.

Finally a more or less painful swelling of doughy or firm consistency develops in rare cases in the region of the ensiform cartilage, behind the left elbow or in the left region of the lower ribs, exceptionally also on more distantly situated places of the body, which breaks open after a time and discharges pus or rather an ill-smelling ichorous fluid mixed with feed material through the opening, and sometimes the foreign body

is also discharged through this opening. The fistula so formed later on sometimes discharges particles of masticated food from the stomach and closes only gradually. It may close after weeks and months and lead to final complete recovery; or a cachectic condition may develop gradually, in consequence of gastric disturbances and extensive adhesions, which finally leads to death.

**Course.** Obstruction of the gastric openings caused by dull bodies is of short duration and either ends in recovery within a few days, that is, if the foreign body is removed either by vomiting or by returning into the interior of the stomach, or death rapidly follows if the obstruction persists. Chronic gastric atony due to dull foreign bodies usually lasts for weeks and months; in the meantime the animals become more or less emaciated or they die within nine to seventeen days if the stomachs are filled with sand or similar material (Krichels).

The inflammation which is caused by pointed or sharp foreign bodies subsides within a few days or weeks if the gastric wall has been only injured but not perforated, or there may be established chronic gastric disturbances which are due to adhesions. If, however, the pointed foreign body has perforated the stomach wall, complete recovery may occur in rare instances if this foreign body slips back into the fore-stomachs or if it pushes itself into the outside world; nevertheless, as a rule the animal exhibits gastric disturbances until death occurs. In certain cases a shorter or longer period of improvement is followed by an aggravation because the foreign body which has slipped back into the stomach again penetrated into the gastric wall. Such variations in the picture of the disease may occur repeatedly. The possibility of recovery seems to be excluded when symptoms appear pointing to inflammation of the neighboring organs. A fatal issue may take place within a few days if the foreign body penetrates rapidly, but the disease usually lasts for weeks and months.

The appearance of complications has always a bad prognostic meaning. Fever likewise is an important factor and a long continued elevation of temperature points to progressive deeply penetrating inflammatory processes.

**Diagnosis.** The exact origin of gastric disturbances due to dull foreign bodies can only be surmised from the history of the case, although sudden bloating excites suspicion as to hair and food balls in the stomach, particularly if the animals have recently shown strong inclination to licking. Exceptionally the correct diagnosis can be made if the foreign bodies are found in vomited material or in the feces. Lodgment of hair balls in the cardia of calves may be assumed, according to Schaubert, if bloating does not yield after the administration of tincture of veratrum (30 to 40 gm. three times daily).



The clinical picture of gastritis due to pointed foreign bodies is characterized by the sudden onset of the gastric disturbances without apparent cause, their variable character, their obstinacy in their further course, tenderness in the region of the ensiform cartilage and in certain cases at the attachment of the diaphragm, and by the deficient use of abdominal pressure. In the absence of the last mentioned symptoms a diagnosis is hardly possible and it can be made only in the further course of the disease, especially if heart symptoms develop and clear up the nature of the disease. In the beginning of the latter an exploratory abdominal puncture (behind the ensiform cartilage between the navel and the milk vein) may perhaps throw light on the case (Sallinger). If the foreign body is still sticking in the gastric wall, or if acute inflammatory processes still exist in the neighborhood of the perforated section of the stomach, the reaction of the fore-stomachs to the administration of motor stimulants is decisive as first pointed out by Johne. If these motor stimulants, causing strong contractions of the stomachs also cause the expression of pain (groaning, restlessness) with an intensification of the gastric disturbances, one is justified in assuming the presence of a traumatic gastritis (Johne, Eber, Holterbach). The absence of signs of pain or the improvement of the condition after the administration of such drugs does not necessarily exclude the existence of gastric disturbances of traumatic origin.

Of the remedies which stimulate the gastric movements tartar emetic or eseridine (see page 254 [Eber]) are particularly to be considered. Holterbach recommends veratrine and arecoline (0.1 gm. in tablets) given in linseed broth alternately every two hours. Veratrine may also be given subcutaneously with advantage, likewise pilocarpine (see page 254).

In differential diagnosis certain cases of overfilling of the rumen, acute bloating and atony of the fore-stomachs due to other causes are to be considered. The important points in differential diagnosis have already been mentioned (see pages 252, 258, 271). General acute peritonitis and gastritis take an acute course with high fever, and grave general disturbances; there are, however, cases in which even these affections cannot be absolutely excluded. Tuberculosis of the lungs can be excluded if certain physical signs which may possibly point to it, are only found in the posterior and lower portions of the thorax, and perhaps only on one side, and if gastric disturbances have preceded the preliminary affection. Sometimes the tuberculin test may decide the question. Tuberculosis of the mediastinal glands can easily be distinguished from traumatic gastritis.—A complicating pericarditis, pneumonia or pleurisy might be confounded with tuberculosis. However, secondary complications of traumatic gastritis are preceded by gastric disturbances, and later they remain in the foreground of the clinical picture. However, all circumstances must be



considered in each case and the possibility of tuberculosis must be kept in mind.

**Treatment.** The same therapeutic methods which are recommended for the cure of overfilling of the rumen (see page 253) should be employed to remove the gastric disturbances caused by dull foreign bodies. In overfilling of the gastrointestinal tract with sand Pötting uses castor oil or mucilaginous solutions, followed after eight hours by opium; he also recommends rhizoma veratri. This treatment is however often unsuccessful in sand in the rumen. After the lodgment of foreign bodies in one of the gastric openings, drugs stimulating the motions of the rumen may also be employed; however, the foreign body can usually be removed only by incision of the rumen. Expectant treatment appears indicated in the beginning since even in the presence of pointed bodies in the stomachs complete recovery is possible; it is then to be recommended to elevate the forepart of the body of the animal (Imminger) and to give them easily digestible or fluid food (see page 273).

The elevation of the foreparts of the body may, according to Sallinger, be accomplished by constructing a bridge 30 cm. high, supplied with cross boards and slanting backwards. The sick animals ought to stand and lie on this improvised bridge for eight or more days.

If emaciation and continuous fever have set in, the animals should be slaughtered unless an operation for the removal of the foreign body has already been decided upon. An incision into the rumen has been performed successfully in the presence of dull foreign bodies (Scherg, Spörer). The incision of the rumen should always be tried in the milder cases and in those not particularly far advanced since the animal may still be slaughtered even if the operation is not a success. However, in long-existing and very grave cases this operation is no longer to be recommended since it will, under these circumstances, not lead to recovery. In some cases the operative treatment of abscesses in the neighborhood of the fore-stomachs has to be considered (Moussu).

The removal of foreign bodies through an incision into the rumen was first proposed by Obich and it was practiced later on by Meyer for the cure of traumatic pericarditis. The operation is performed by making an incision into the left flank of the standing animal (see page 255). The hand is then introduced through the operation wound and guided towards the opening between rumen and reticulum, situated to the right and below. If the foreign body is found it is extracted. Obich was successful in four out of thirteen cases and Meyer caused in one case the disappearance of the pericardial symptoms which were already developing.

Schöberl's method of removing a foreign body which had penetrated into the gastric wall by massage, consists in placing the animal on its back. The operator then stands next to the animal on a chair and with one foot placed upon the ensiform cartilage he pushes upon it; six to ten times is usually sufficient. It is claimed that in only two out of sixty animals so treated did the foreign body fail to slip into the reticulum. Considering the fact that a sudden increase of

intra-abdominal pressure may favor the penetration of the foreign body into one of the neighboring organs, the procedure of Schöberl appears to be not without danger in many cases, in spite of the fact that Schöberl and also Estor claim to have had such good success. The numerous reported recoveries admit of the probability that the procedure has also been employed in many cases which were not traumatic gastritis.

**Prophylaxis.** The number of cases of gastric disturbances due to foreign bodies may be considerably diminished by proper instruction of owners and attendants of animals. Harms recommends the construction of a sink-hole at the end of the crib for the reception of foreign bodies. Rust recommends for neighborhoods where hay frequently contains iron foreign bodies that the chaff cutter should be supplied with a magnet which removes iron particles. The swallowing of hair and wool may be prevented by curing the habit of greedy licking or the itching skin affections, by constant supervision of the animals, and by separating the lambs from the ewes.

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**Other Inflammatory Diseases of the Fore-stomachs.** (Ruminitis Reticulitis, Omasitis.) Aside from traumatic gastritis, primary inflammation of the fore-stomachs is very rare as, for instance, after the ingestion of hot liquids (distillers mash), irritating plants or caustic drugs, also under the influence of an external traumatic injury acting upon the fore-stomachs. Dieckerhoff saw in three head of cattle a diphtheritic inflammation of the fore-stomachs and of the esophagus; since the diphtheritic foci looked very similar to trichophyton infection in cattle, the observer considered it possible that the affected animals might have ingested the mould by licking animals affected with this skin disease. In rare cases diphtheritic necrotic masses of roundish outlines are found in the rumen, in the reticulum or the omasum, also possibly in the abomasum, which have a diameter of 1 to 7 cm. After the shedding of the dead parts, the individual leaves of the manyplies have a fenestrated appearance or their margins become irregular in outline. The crusts have been seen up to 2 cm. thick, dry, fissured and dark gray; they sometimes possess a central area of softening. Complete healing may occur in these cases with the formation of radiating cicatrices in the rumen and with permanent fenestration of the leaves

of the omasum. The cause of these necrotic processes is probably a superficial invasion by the bacillus necrophorus.

The inflammation of the fore-stomachs is usually a secondary affection, appearing during the course of certain infectious diseases (foot-and-mouth disease, rinderpest, malignant catarrhal fever, sheep pox); also after gastric atony (see page 265) or overfilling of the rumen (see page 249). Tuberculous ulcers in the omasum have been described by Johne and Weiser.

The **symptoms** of inflammation of the fore-stomachs are very similar to those of gastric atony, but with that difference that repeated vomiting occurs; there is a diffuse tenderness of the fore-stomachs, and from the start a moderate fever. The rumen does not feel hard as in cases of atony. Appetite and rumination are suppressed partially, or often entirely, while thirst is increased. If there is a concomitant inflammation of the esophagus there is difficulty in deglutition.

In the majority of cases the animals recover within eight to ten days. But if the inflammation has penetrated more deeply, there is diarrhea; the pulse-beat rises to from 80 to 100 and the animals succumb after two to three weeks from exhaustion or peritonitis.

The **treatment** is similar to that in atony of the fore-stomachs (see page 273); it is however not very promising in inflammations which penetrate more deeply or in those of the diphtheritic type.

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### 3. Acute Catarrh of the Stomach. Catarrhus ventriculi acutus.

(*Gastricismus, Gastrosis, Status gastricus, Dyspepsia acuta, Febris gastrica.*)

**Etiology.** Acute catarrh of the stomach, including catarrh of the abomasum in ruminants, occurs either as a primary affection per se or as a secondary complication due to other organic diseases or as part of the picture in the course of general diseases.

The most common cause of **primary catarrh** of the stomach lies without doubt in errors of diet, if an otherwise unimpeachable food material gets into the stomach without the proper preparation, in improper quantities or at irregular intervals. Starved animals go greedily for feed and try to satisfy their hunger rapidly; they do not masticate properly and do not mix the food sufficiently with saliva, hence the feed enters the stomach insufficiently broken up, in coarse masses and half dry. This irritates the gastric mucosa mechanically and the digestion of the food is insufficient. For these reasons dogs and swine are more often affected than horses; gastric catarrh is particularly common in animals which are fed only once a day. In cattle, however, the excessive ingestion of water, especially in summer, may cause catarrh of the abomasum.



Irregularity in feeding has likewise a detrimental effect, not only because the animals feed too greedily after starvation, but also because a stomach used to periodical function will easily become disturbed in the secretion of the gastric juice, if food is not forthcoming regularly. For this reason livery stable horses, which cannot always be fed regularly, suffer frequently from the disease.

Disturbances in mastication may indirectly cause gastric catarrh; this occurs in tenderness of the gums during the change of the teeth, in anomalies of the teeth, such as are seen frequently in horses, in wolf-teeth, in the different types of stomatitis with a profuse secretion of a subsequently decomposing saliva. All these factors may be detrimental to the gastric mucosa. Disturbances of rumination exert a harmful influence upon the function of the abomasum in ruminants.

Overloading of the stomach with large quantities of food which is otherwise unobjectionable occurs preferably in carnivora and hogs, because these animals are often too greedy in the ingestion of food. But horses likewise suffer from this disease if, after rather poor dry feed, they immediately receive larger amounts of palatable fresh feed, or if they can pasture at random in luxuriant clover or alfalfa fields, or if horses which have gotten loose in their barn can get into the feed supplies and can there eat as much as they want. In such cases the feed mash ferments and the gastric mucosa is irritated by butyric, lactic and acetic acid. In ruminants catarrh of the abomasum occurs through the same irritation. Starving for long periods likewise leads to catarrh. Andral and Gavaret have found catarrhal changes upon postmortem examination of starved dogs.

Improper condition of the feed may also lead to catarrh of the stomach, as, for instance, the ingestion of too cold or too hot food, such as hot distillers' mash, frozen potatoes or beets, frosted grass, ice cold water. Hot medicinal administrations may have a similar effect.

Spoiled feed is still more harmful, especially if given exclusively or mixed with comparatively little good feed. Spoiled feeds are mouldy, sloppy, marsh-hay, mouldy cereals, moist decaying straw, intensely fermenting green feed, rotten potatoes and beets, decomposed rancid milk, fermenting old garbage, etc., and dirty foul stagnant water.

A catarrh of the stomach which is more or less severe may also be brought about if the feed contains acrid and irritating plants or food stuffs which are strongly seasoned or mixed with fragments of bone. Acrid and cauterizing drugs have a similar effect if given in a large single dose or if administered repeatedly in smaller doses, for instance, the drastic laxatives, tartar emetic, calomel, arsenic, acids and alkalies, etc.

Food of improper composition often leads to catarrh of the stomach in cattle, for instance, overfeeding with distillers'

mash, germinated malt, beets and bran, potatoes, household garbage, factory remnants, leaves, bulbs. Exclusive feeding on straw likewise is often followed by gastric catarrh. Similar errors of diet in horses are more rarely observed, but they occur if these animals are fed with corn, rye, bran, crushed grain, and leguminosæ, without giving them time to adapt themselves to such food stuffs. Very young oats and insufficiently dried hay are usually also detrimental. Hogs suffer particularly if fed too much kitchen garbage. Foreign bodies in the feed are rarely of importance.

One must look for an infection if the gastric catarrh appears simultaneously among several animals without any apparent cause and with febrile symptoms. This type is most commonly seen in horses; its cause, however is not as yet known.

Animal parasites may likewise cause catarrh of the stomach if present in larger numbers.

Of external factors over-exertion may bring about catarrh of the stomach, partly in consequence of congestion of the peripheral blood vessels, partly if by reflex irritation the stomach is prevented from discharging its contents.

The different noxious factors do not always influence animals in the same way; they are frequently borne without harm and particularly in animals which are not so well attended and consequently more hardened. This individual variability in resistance must be considered, and the same method of feeding may be harmful to some individual animals and perfectly harmless to others.

**Secondary acute catarrh of the stomach** frequently accompanies acute infectious diseases and quite commonly constitutes their earliest manifestation. It is also seen in chronic exhausting diseases such as chronic diseases of the heart, lung, and liver, where venous congestion leads to catarrh of the stomach. Catarrh of the abomasum is a frequent complication of disease of the fore-stomachs in ruminants.

**Anatomical Changes.** Intense reddening, serous infiltration, loosening of the tissues of the mucosa, the presence of an abundant tenacious, glairy, sometimes more or less purulent mucus are the most marked anatomical changes. The mucosa is thrown into thick rugæ, which show reddening on their free margins, or even more extensively, and also occasional small hemorrhages and superficial erosions. These changes are most marked at the pylorus and even in such animals where the whole stomach is provided with a glandular mucosa.

In interpreting the post-mortem findings, one must always remember that certain changes which existed during life, particularly hyperemia, may disappear post mortem and that the gastric mucosa always contains an excess of blood during digestion.

**Symptoms.** The most important symptom in catarrh of the stomach in all animals is a decrease of appetite, and often its



entire suppression. The animals approach the feed offered without appetite, they eat little and slowly, soon stop or they refuse the ingestion of food entirely. Sometimes the animal greedily licks unpalatable, undigestible, bitter, repulsive materials, or they accept only certain kinds of food. Frequently the appetite is changeable; some meals are taken in a normal manner, then again there is an entire absence of appetite. Thirst is either diminished, or on the contrary increased, particularly if vomiting exists; some animals take only fresh cool water, others again stale water or water contaminated by manure.

Another common symptom is dullness of the sensorium. The animals are indifferent to their surroundings, are feeble, apathic, stand or lie quietly in one place, do not like to move and rapidly tire at work.

The temperature is either quite normal, or perhaps there is a very insignificant elevation; the surface heat is distributed unequally. Cases which set in with high fever are rare, and even in these the fever subsides rapidly provided there are no complications.

The other symptoms vary more or less according to the various species of animals.

In the **horse** one frequently observes gaping, the animals lick iron and other cool objects and nibble on the walls. (These abnormal desires are probably due to abnormal stimuli upon the terminal nerve fibers of the pneumogastric nerve.) There is occasionally belching or even vomiting, especially if the feed had contained intensely irritating substances; such rare symptoms are however commonly seen more in dilatation or grave inflammation of the stomach.

Mucus and saliva collect abundantly in the buccal cavity forming a sticky, tenacious deposit upon the mucosa and there may even be symptoms of stomatitis with furred tongue and fetid breath.

In consequence of the diminished ingestion of food, and also from reflex effects, the intestinal movements are retarded. The abdomen becomes somewhat extended, intestinal sounds become less frequent, defecation is retarded, the feces often are discharged as small dry balls covered with a shiny coating and they contain undigested food particles. The amount of urine is, as a rule, diminished.

Since catarrh often extends to the duodenum there is frequently an icteric discoloration of the mucosæ.

**Cattle** appear markedly feeble; they stand with their backs curved, draw back from the crib, the skin is coarse, the ears are dropped and exceptionally they present the symptoms of dull abdominal pain. Detroye states that in exceptional cases there are signs of agitation. The muzzle appears moist, but if rubbed dry it takes a longer time until it becomes again covered



with drops of moisture. The conjunctivæ are usually reddened and frequently show an icteric discoloration. The pulse is accelerated, small, in grave cases it cannot be felt at all. The secretion of milk suddenly becomes decreased to one half and becomes entirely suppressed in the further course.

Rumination is sluggish, infrequent, irregular or entirely absent. Disagreeable fetid gases smelling, according to some observers, like onions are expelled by belching. The region of the left flank appears either deeply depressed or somewhat more tense, but without being bloated; however, there is occasionally some bloating of the rumen in the later course of the disease, because somewhat more deeply penetrating catarrhs produce atony of the fore-stomachs. The motions of the rumen are somewhat less frequent, less energetic, but they are normal as soon as the rumen is full (Harms). Under the right costal arch, in the region of the abomasum, there is tenderness on pressure. Defecation occurs less frequently than under normal conditions; the feces are dry, dark in color, cylindrical, and they are alkaline in reaction (Harms), sometimes covered with a crust and containing coarse, undigested parts of feed. If extensive intestinal catarrh follows upon catarrh of the abomasum, there exists diarrhea.

In sheep and goats the symptoms are similar to those in cattle. The sick animals separate themselves from the herd and stand with head low or lie down.

In **carnivora** vomiting is one of the most important symptoms and it is absent only in very mild cases. After overloading their stomachs, these animals vomit the ingested feed in large quantities mixed with saliva; after this their condition improves markedly, and they recover soon. In other cases vomiting only occurs directly after the ingestion of food and water, rarely independently of it; in these cases the vomited matter consists frequently of only tenacious, glairy slime, possibly mixed with streaks of blood; if the vomiting occurs very frequently bile is also present and the masses are tinged with green.

The tongue is coated and dry, the smell from the mouth is occasionally repulsive, thirst is increased as a rule, the region of the stomach under the left costal arch or behind the ensiform cartilage is tender to pressure. Defecation is retarded, at first dry, later on soft and fetid; on standing the urine forms a deposit of urates (*sedimentum lateritium*).

In **hogs** the disease takes a course similar to that in **carnivora**; the unequal distribution of temperature over the body may be recognized by the fact that the ears and extremities are cool, the nose however being warm to the touch. The sick animals drop their tails straight down, burrow under their straw and drink much water.

In **rabbits** one observes, aside from the lack of appetite, abnormal desires like licking and chewing of the walls, inges-

tion of wet straw or manure, occasionally also vomiting. The abdomen appears somewhat bloated and is tender to pressure. Defecation is retarded, the feces are dry and dark in color, and diarrhea occurs sometimes in the further course. There is sometimes also an icteric discoloration of the conjunctivæ (Braun).

**Course and Prognosis.** Primary acute catarrh of the stomach usually runs its course in a few days, at the utmost in 1 to 2 weeks, and ends in complete recovery. However it is not advisable to make an absolutely favorable prognosis from the start, because catarrh may lead to gastritis and it may frequently be followed by catarrh of the intestines, which is of considerable importance in young and also in very old animals. Moreover catarrh may become chronic and consequently more obstinate.

The importance of secondary acute catarrh depends chiefly upon the nature of the underlying primary affection and it influences the latter unfavorably.

**Diagnosis.** Primary acute gastric catarrh can easily be recognized since the history of the case frequently furnishes sufficient data. It is however somewhat difficult to decide whether catarrh is confined exclusively to the stomach or whether it has already extended to the intestines. The signs which are of significance in the differential diagnosis will be fully considered under intestinal catarrh, but it ought to be pointed out here that gastric catarrh as a rule is accompanied by catarrh of the duodenum, which manifests itself in an icteric discoloration of the mucous membranes. Abdominal pain which can occasionally be observed points to a simultaneous catarrh of the intestines. Gastric catarrh is frequently accompanied by a similar affection of the intestines and in this case the disease develops into a gastro-intestinal catarrh, and it is then difficult to analyze the observable symptoms for differentiation. Intestinal sounds, the condition of the feces, and the presence or absence of icterus may however furnish enlightenment.

In ruminants it is often difficult to distinguish gastric catarrh from affections of the fore-stomachs, because the latter make it impossible to recognize whether the abomasum is intact or not, and affections of the fore-stomachs may be followed by disease of the abomasum. If there is localized tenderness upon pressure in the region of the former and an icteric discoloration of the conjunctivæ, if the left flank is fallen in or at least the rumen not full, and if further the movements of the rumen are not markedly diminished, catarrh of the abomasum may be assumed to exist. Sometimes a history of errors in diet will furnish valuable data.

The gastritis is distinguished from simple catarrh by the greater intensity of the affection and its symptoms, by the great prostration, by the frequent and weak pulse; traumatic gastritis of cattle can be recognized by a distinct tenderness upon pressure in the neighborhood of the omasum.

**Treatment.** The first object of treatment is a proper consideration of the affected gastric mucosa in order to protect it against further injury. It is therefore advantageous, particularly in overfilling of the stomach, to starve the animals for a few days or to give them very little of an easily digestible food. Herbivora may have a little fresh green feed, bulbous plants, oat straw, thin-fluid salted meal or bran, or oat-meal, barley-meal, linseed-cakes; hogs may be given barley-corn or sago-soup, oat-meal with some fat and salt, scraped meat; valuable dogs may have meat-peptone as a broth in tablespoonful doses, somatose (1-3 teaspoonfuls rubbed up with broth); plasmon boiled and added in tablespoonful doses to soups or milk; robo-rat (2-4 tablespoons in feed or soup), tropon (like the former). The ingestion of water must be restricted as much as possible. In ruminants, however, abundant drinking water is frequently beneficial; to stimulate thirst the animals may be permitted to lick salt or they may receive mealy mash which are strongly salted.

In overloading of the stomach in hogs and carnivora the use of emetics is indicated. The best drug is hydrochlorate of apomorphine (for dogs 0.005-0.01 gm.; for cats 0.002-0.005 gm.) or veratrine (for hogs 0.003 gm.—injected subcutaneously. Less advisable is the administration per mouth as for instance root of ipecacuanha (for hogs 2-3 gm.; for dogs 0.5-2.0 gm. or rhiz. *veratri albi* (0.5-2.0 or 0.05-0.2 gm.). Hogs may also be treated by a rectal injection of the decoctum *veratri* (2.0 to 50). Frequently the introduction of warm water or warm salt solution into the stomach will cause vomiting.

Washing out of the stomach (gastric lavage), may easily be practiced in carnivora and even in horses and young pigs and it is to be recommended in over-loading of the stomach.

In horses and cattle one should attempt to empty the stomach contents into the intestines by the administration of mild laxatives such as salts, tartar emetic with sulphate of sodium or magnesium (for horses 3.0 to 100.0 grammes; for cattle 4.0-6.0 gm. to 500-800 gm. in two doses). For smaller animals castor oil is indicated (for calves, foals, sheep, goats 50.0-200 gm.; for hogs 30-100 gm., for dogs 15.0-60 gm., for cats 5-20 gm.) or calomel (according to Müller for dogs 0.3-0.4 gm.; for cats 0.1-0.15 gm.). If there is obstinate vomiting dogs should have cracked ice, water containing bicarbonate of sodium, or champagne, or opium (0.05-0.10 gm.) or other narcotics.

To remove digestive disturbances, hydrochloric acid is most valuable; it is best added to the drinking water (for horses and cattle 10-20 gm., sheep, calves and hogs 2-5 gm., dogs 0.2-0.5 gm. *pro dosi*). Very serviceable, particularly in horses, are artificial Carlsbad salt and laxative salts in general (horses up to 50-80.0 gm.; cattle 200.0-250.0 gm. in mucilaginous media) or combined with bitters (*gentiana*, *calamus*, *rheum*, etc.). Smaller animals may receive pepsin (0.1-0.5 gm.) perhaps in combination



with hydrochloric acid (0.1-0.5 gm. in water); also the various bitter tinctures (Tr. rhei vinosa et aquæosa,—tinct. gentianæ, tinct. chinæ composit., tinct. amara, vinum chinæ, vinum condurango 5-15.0 gm.). *Orexinum tannicum* may be given as an appetizer for dogs in doses of 0.1-0.4 gm. in broth.

**Literature.** Berner, D. t. W., 1894, 140.—Dieckerhoff, Spez. Path., 1892, II, 464.—Eber, Z. f. Tm., 1906, X, 321.—Ellenberger & Hofmeister, A. f. Tk., 1888, XIV, 55.—Harms, Rinderkrkh., 1890, 56, 65.—Hübner, S. B., 1888, 65.—Imminger, W. f. Tk., 1907, 1.—Jacob, W. f. Tk., 1908, 105, 107.—Leisering, S. B., 1876, 78.—Moussu, Maladies du bétail, 1906, 229.—Schlampp, Ther. Technik, 1907, 86.

#### 4. Chronic Gastric Catarrh. *Catarrhus ventriculi chronicus.*

(*Gastritis catarrhalis chronica.*)

**Etiology.** The various detrimental factors which cause acute gastric catarrh (see page 285) also play a rôle in the production of chronic gastric catarrh, if they act over longer periods, or if acute catarrh recurs frequently in short intervals. It is therefore not necessary to refer again to the causes and it may be sufficient to point out, that the majority of cases of chronic gastric catarrh stand in close causative connection to the ingestion of spoiled feed, to mastication interfered with by diseases of the mouth and teeth, or to excessive overwork.

Further causes are foods which produce a relaxation of the gastric wall with subsequent dilatation, so that the feed is mixed less intimately with the gastric juices, and this again leads to the formation of fermentation products which irritate the mucosa. Such food stuffs are withered or frozen green feed or bulbous plants, very dry bran or meal, warm distillers' mash (alcohol here also is a causative factor of catarrhal conditions), if these are given with little straw or hay, also germinated heads of rye.

The ingestion of sand with food or water causes a chronic gastro-intestinal catarrh in horses, as is frequently seen on the Kirgisian pastures in Siberia; the Kirgisians call this disease "Kum-Gata" (sand-disease). Sand-disease in buffaloes and cattle has previously been mentioned on page 276 (Marccone, Müller, Prietsch).

Every congestion of the portal system, no matter to what disease it may be due (diseases of the liver, lungs, heart), extends into the gastric mucosa. Chronic passive congestion of the gastric mucosa leads to a condition which is identical with chronic catarrh. (What Graf described as an edema of the abomasum and some of the cases reported by Harms as chronic gastro-intestinal catarrh of cattle were very probably nothing else but an enteritis paratuberculosa or Johne's disease.)

In horses chronic gastric catarrh is sometimes due to the swallowing of air, causing dilatation and diminished contractibility.

**Chronic gastric catarrh** in general may be brought about by all those factors which may influence the motility of the stomach unfavorably. Hence the disease is common in older animals, when their gastric function has become insufficient, particularly if such animals ingest too much food.

Animal parasites are a frequent cause of chronic gastric catarrh (see gastric diseases due to worms).

**Anatomical Changes.** The mucosa of the stomach, particularly in the region of the pylorus, is covered with a turbid gray, tenacious and firmly adherent mucus, which is occasionally more like pus or mixed with blood. The mucosa itself is more or less thickened, corrugated either uniformly or only in spots; it is brownish red, especially at the apex of the rugæ, and it is slate-gray in color in advanced cases. In a horse dead from exhaustion the wall of the stomach was found to be three times the normal thickness; the mucosa of the stomach and of the duodenum was of a lardaceous appearance (Pr. Mil. Vb., 1907). More rarely the mucosa is pale and thin. In consequence of unequal regeneration of tissue the surface of the mucosa becomes peculiarly nodular with prominent polypoid excrescences, especially between the openings of the glands (gastritis prolifera s. verrucosa s. polyposa). Subsequently a dilatation of the stomach usually occurs.

**Symptoms.** In the horse chronic gastric catarrh usually manifests itself by a diminished appetite. The animals feed very little or not at all. This variability of appetite is occasionally accompanied by morbid desires, causing the animals to ingest alkaline, bitter and also completely indigestible materials.

The animals are dull and tire easily at work. If the catarrh has existed for some time, emaciation occurs, particularly if the animals have been overworked during this period. The mucosæ become pale or yellowish, the abdomen becomes drawn in, the hair coat appears lusterless and rough, the skin non-elastic and sometimes affected with eczema. In some cases the patients also show signs of abdominal pain.

The tongue appears coated, a tenacious, sticky, sweetish-smelling saliva frequently collects in the mouth. Defecation is retarded, the manure is dry and contains undigested food particles; occasionally there is also light diarrhea. The urine is scanty and forms a considerable sediment on standing.

Nervous disturbances may occasionally become manifest. The dullness increases to such an extent that it approaches dumb-staggers, and there may be disturbances of locomotion as in vertigo (vertigo abdominalis).

In the "Kum-Gata" disease of horses (the Siberian sand disease) one observes languor, feebleness, lack of appetite, increased thirst, con-



stipation, polyuria and paleness of the mucous membranes. Pulse and respiration are increased and there is some fever ( $38.5-39.5^{\circ}$  C.). In the further course constipation and diarrhea alternate, the feces are grayish-green and very ill smelling. The emaciation progresses rapidly and edematous infiltration of the subcutaneous connective tissue sets in. The disease leads to a fatal issue within three to four weeks.

In **ruminants** sand disease of the stomach often leads to symptoms similar to those in the horse. In other cases of primary catarrh of the abomasum, which is comparatively rare, one sees diminished appetite, retarded mastication and morbid desires. In consequence of the subsequent development of a chronic intestinal catarrh there is occasionally diarrhea, alternating at variable intervals with constipation; after several weeks, however, diarrhea is the more prominent symptom. The secretion of milk becomes entirely suppressed, emaciation and cachexia become more marked and the animal finally dies after the disease has lasted several weeks or months.

Chronic catarrh of **carnivora** hardly presents any other symptoms except variability of appetite and nutritive disturbances. The region of the stomach may be somewhat tender to pressure; vomiting is rare and usually occurs immediately after the ingestion of food. The patients vomit a tenacious mucus mixed with food particles.

**Course and Prognosis.** The course of chronic gastric catarrh depends largely upon its cause. If it has developed in connection with another chronic affection it is usually of grave importance because it cannot be easily cured and causes in itself a considerable ill effect upon the underlying condition. Even in primary cases of chronic gastric catarrh the prognosis is not favorable, because permanent tissue changes have usually been produced and these cannot be made to disappear. The prognosis also depends upon our ability to stop the detrimental effects of gastric disturbances by the administration of easily digestible food.

**Diagnosis.** The diagnosis of chronic gastric catarrh is difficult since the symptoms are usually not characteristic and since similar symptoms occur with or without gastric catarrh in various chronic diseases (particularly in diseases of the liver). A diagnosis of gastric catarrh may, therefore, be made after the exclusion of other diseases which lead to similar disturbances of nutrition. If it is justifiable to assume the presence of chronic gastric catarrh, it is next desirable to find out whether the affection is primary or due to other diseases of the stomach (tumor, ulcer, animal parasites).

Among the symptoms, the most important are variability of appetite without any apparent external cause, a morbidly changed sense of taste, and in carnivora vomiting after the in-



gestion of food. The diagnosis may be assisted by ascertaining whether any errors in diet have been committed.

**Treatment.** The proper regulation of diet is of much more importance in the treatment of chronic gastric catarrh than the administration of medicines. By properly selected food, given in a proper manner, it is indeed frequently possible to cure digestive disturbances or at least to keep the animals in a fair state of nutrition, while the administration of medicines alone has very little value and an often repeated medication will frequently make the condition worse.

Those food stuffs are best which are juicy, soft, mashy in consistency, since they remain in the stomach a comparatively short time and are easily passed into the intestines. Herbivora do best with fresh green feed, soft hay, oats, straw not chopped too short with some oats, bran or oat-cuttings, also bulbous plants; the cereals which are more difficult to digest are less to be recommended; if they cannot be avoided, however, they ought to be superficially roasted or their flour ought to be first macerated at body temperature. In some cases preparations containing diastase, such as diastasolin or diafarin may be administered with floury suspensions. (According to Schade a horse ought to receive one-half pound of oat-flour stirred with one quart of cold water; this is then poured into two quarts of boiling water; when the starchy emulsion so prepared has cooled down to 55° C., 13 gm. diastolin, previously stirred up in a cupful of lukewarm water, is added; the saccharification of the starch is complete in twenty to thirty minutes. In carnivora one may advantageously give meat broth with the yolk of eggs, finely chopped and salted meat of young animals, also milk-stews, etc. All conditions which prevent proper mastication must be looked after; the improvement of the condition of the teeth in older horses frequently leads to long-lasting improvement of digestion.

Drugs should be administered only in combination with the proper food, and as a measure to support the dietetic régime. Hydrochloric acid does very well in small doses (for large animals 10.0-15.0 gm., for small ones 0.5-1.0 gm.) added to the drinking water or given very much diluted (1:250 may be given as a draught); such doses may be continued for a considerable time. Their effect may be increased by the addition of pepsin (5-10 gm. for large or 0.1-1 gm. for small animals). The neutral salts are to be recommended; herbivora may have access to rock salt, so that they can lick it when they like, or the salt may be powdered and added to the feed. Artificial Carlsbad salt has a good effect; it is mixed with the food for horses (30-50.0 gm.) or given in the form of pills to ruminants (50-80 gm.) or it may be given for weeks, dissolved in water one-half to one hour before ingestion of food. In order to stimulate the secretion of the peptic glands, bitters may be added

to the neutral salts (gentiana, calamus, rheum). Harms recommended in chronic gastric catarrh of cattle the continuous administration of Carlsbad salt in increasing doses daily from 50-150 grammes; the dose dissolved in a bucketful of water and given one hour before the early morning feeding.

If in ruminants the symptoms also point to serious disturbance in the function of the fore-stomachs, the treatment used in atony of these stomachs appears indicated (see page 273).

In carnivora it is advisable to add to the regulation of the diet the administration of hydrochloric acid (5-10 drops in 250 cc. shortly after the ingestion of food); also bitters (especially the bitter tinctures in teaspoonful doses, decoction of cortex quiniæ, of colombo- or of condurango-root); also neutral salts, preferably Carlsbad salt (one teaspoonful daily in warm water upon an empty stomach). If there is much fermentation, salicylic acid (0.2-0.5 gm. before feeding), resorcin (0.1-0.2 gm.), creosote (0.1 gm.), naphthalin (0.1-0.3 gm.) in several repeated doses may be of advantage.

**Literature.** Ellenberger & Hofmeister, A. f. Tk., 1888, XIV, 55.—Petit, Germain & Breton, Bull., 1907, 428.—Wedernikow, Vet. Jhb., 1894, 84.

## 5. Acute Dilatation of the Stomach. *Dilatatio ventriculi acuta.*

### (a) Acute Dilatation of the Stomach of the Horse.

(*Magenüberladung, Magenüberfüllung, Ueberfütterungs-Kolik*  
[German]; *Colica crapulosa; Indigestion stomacale*  
[French].)

Acute dilatation of the stomach of the horse is either caused by the ingestion of unusually large masses of food, or by an impediment to the emptying of the stomach. The heretofore normal stomach becomes dilated, but if the cause is removed it again assumes its normal volume.

**Occurrence.** This disease, known as overloading of the stomach, overfilling of the stomach or overfeeding colic, occurs particularly in horses which receive improper food or are fed at irregular intervals, or which are exposed to excessive exertion. This explains why the disease occurs so frequently in large cities, where the attendance, the feeding, the working of the horses is improper in many respects. The disease is rare in horses kept as breeding animals, in those kept for pleasure, or in farm animals. The affection also occurs among army horses, especially during camping and maneuvers, on account of the prevailing irregularity in feeding.

In the clinic at Budapest acute dilatation of the stomach is seen annually in 11-21% of the horses sick with colic; this is a percentage probably not markedly different from that in other large cities. The frequency of the disease varies with the season, since it is twice as prevalent during the second and third quarter as during the other quarters of the year. Most cases are seen in July and August.

**Etiology.** **Primary acute dilatation of the stomach** may be caused especially by the ingestion of unusually large amounts of feed (ten quarts or more of oats or other feed). The stomach cannot keep in motion and intimately mix an excessively large mass of feed with the proper amount of gastric juice, hence the feed remaining longer in the stomach swells and leads to the formation of large amounts of gas. The disease also occasionally appears after the ingestion of fresh hay or after-math, or after pasturing on luxuriant pastures.

In the larger number of the cases the cause lies, however, not in the ingestion of too large amounts of feed, but in feed of improper quality; we particularly must mention in this respect indigestible corn, corn-ears, rye, marshy hay, coarse straw, feed which swells easily (bran, peas, beans) or easily form conglomerations (short-cut chaff with corn or barley-meal or bran) or food which ferments easily (young, withered clover, alfalfa, molasses, esparsette, beets and potatoes, grass heated by fermentation and green feed in general, especially if the animals drink much water after its ingestion.) Spoiled feed (moist, mildewy oats or hay or rough feed, mash) may have a similar effect. Improper food is particularly dangerous when the horse has no chance to become used to it gradually, that is, if there is a very sudden change of feed. Improper food is the more liable to produce dilatation of the stomach the more of it is ingested. The affection develops exceptionally in connection with the ingestion of too cold water (Mouquet).

Overexertion or heavy work immediately after feeding also plays an important rôle in the production of the disease. Even proper and unobjectionable feed may cause dilatation of the stomach if the horse is used for heavy work immediately after feeding. This fact, known from actual observation, has been explained by the experiments of Tangl and Scheune which show that physical exercise prevents the emptying of the horse's stomach and leads to a brisk excretion of water into the stomach.

Certain anomalies of the teeth, which prevent the breaking up of the food stuffs, without however causing a diminution of ingestion, sometimes also cause the insufficiently masticated feed to remain in the stomach for a longer time and to ferment.

The fact that some horses are more predisposed to the affection than others may be explained by a variability in the functional capacity of the gastric muscularis.

Dilatation of the stomach is frequently seen as a **secondary affection** to certain intestinal diseases. Obstruction of the small intestines leads the more rapidly to dilatation of the stomach the nearer the obstruction is to the stomach. Not uncommonly dilatation of the stomach is secondary to displacements, obstruction and bloating of the large intestine. The causes of such secondary dilatations vary. Antiperistalsis occurring on various occasions may force the contents of the intestines towards



the stomach; a serious affection of the intestines also prevents the evacuation of the stomach by reflex irritation; besides every somewhat intense increase in the size of the stomach-like dilatation of the colon prevents mechanically the flow of the gastric contents towards the small intestines, because the pylorus is pressed against the stomach by the dilated and tense large intestine. Secondary gastric dilatation occurs more easily the sooner the causative affection sets in after the ingestion of food.

**Pathogenesis.** The causes of acute dilatation of the stomach bring about swelling of the food contained in the stomach. The gastric contents then ferment in consequence of insufficient mixing or of inherent fermentability so that more or less gas is formed. The gastric mucosa is stimulated to an increased secretion and all of these factors lead to a stretching and dilatation of the stomach. Whether after ingestion of coarse or of poorly masticated food a tonic contraction of the pylorus occurs by reflex irritation has not been proven, but neither has its occurrence been excluded. The rapidly developing tension of the gastric wall and the irritation of the mucosa by the decomposition products of feed mash produce convulsive contractions of the gastric muscularis; these cause colicky pains, which are quite intense since the contractions are severe, recur frequently and last a long time. They are, however, not intensified by a sudden increase of intraabdominal pressure as in throwing down or rolling. The drier the contents of the stomach, the less gases or irritating decomposition products are formed and the less energetic are the contractions of the stomach; these muscular contractions may not appear at all after the ingestion of cold water, which acts as a paralyzant to the muscularis. The stomach pushes the diaphragm forward proportionately to its size, making respiration more difficult; however, in consequence of the diminution in the intra-thoracic negative pressure and on account of the mechanical effect against the diastole of the heart, this organ receives less venous blood, and the heart beat usually becomes more rapid. In dilatation of a high degree the internal tension can become so great that the wall of the stomach ruptures. Considerable losses of substance of the gastric wall (caused by the presence of *gastrophilus* larvæ, gastric ulcers) predispose to this accident. However, rupture is probably more frequently due to a sudden increase in the intraabdominal pressure, as it occurs in throwing down, in reckless rolling and vomiting. It can also well be imagined that occasionally rupture may occur in consequence of violent contractions of the gastric muscularis with an intense increase of intragastric pressure.

Dassonville and Brocq-Rousseau experimented on the normal stomach of dead horses and found that the introduction of 26-40 liters of air or of 10-17 liters of water were sufficient to cause rupture of the gastric wall; a manometer connected with the pylorus showed a pressure of 5 cm. mercury only. Fayet and Gattend

found an interior pressure of  $1/12$  atmosphere. Marek made his experiments by connecting the manometer with the pylorus by means of a rubber tube filled with water and he found that the normal gastric wall of the dead horse required an interior pressure of 10. to 13.5 cm. of mercury before rupture occurred.

**Anatomical Changes.** The stomach is dilated to a multiple of its volume; after a cut into the tense gastric wall large amounts of gases escape often in a forcible manner. In rare cases the stomach contains only food-mash and gases; usually there is much fluid mixed with the food-mash. The mucosa shows evidence of an acute catarrh, sometimes also hemorrhages. In rupture of the stomach (*ruptura ventriculi*) one finds usually gastric contents, more or less mixed with blood, in the anterior portion of the abdominal cavity or between the fold of the omentum majus; the peritoneum shows the signs of an acute inflammation. Rupture occurs always in the region of the large curvature (in the experiments of the authors, mentioned above, the rupture was likewise in the same region); the margins of the tear are swollen, ragged, covered with blood coagula, the opening is largest in the peritoneal coat, smaller in the retracted muscularis, smallest in the mucosa. Sometimes the peritoneal covering of the stomach alone ruptures, while the two other strata of the wall remain intact. In other cases muscle fibers in the neighborhood of the cardiac end are pushed apart so that the mucosa here rests directly upon the peritoneum.

The lungs are in a condition of passive congestion; sometimes there is a tear into the diaphragm with a protrusion of parts of the intestines or liver into the thoracic cavity.

**Symptoms.** The symptoms of primary acute dilatation of the stomach usually occur within four, exceptionally only seven hours, after ingestion of feed. The patients draw back from the crib, lie back in their halter, and are listless. Very soon the symptoms of colic appear; these are severe and protracted or they may be interrupted by short, painless intervals. Sometimes such intervals are entirely absent. The animals throw themselves recklessly, roll persistently and often sit down on their haunches in dog-position. However, occasionally the colicky symptoms are only mild; they are entirely absent in dilatation of the stomach due to the ingestion of cold water (Mouquet). In such cases the authors have, however, always found on post-mortem examination an inflammation of the gastric mucosa. The claim of Forssell that violent symptoms of pain are absent in uncomplicated dilatation of the stomach is disproved by general experience.

The exhaled air frequently has a peculiar sour smell. Belching occurs in the majority of cases, sometimes also gagging and vomiting, with the expulsion of a sour-smelling fluid mixed with particles of food from the anterior nares and also sometimes from the mouth. One can also observe undulatory motions towards the head along the jugular groove. Pressure upon the left jugular groove often excites belching.

The stomach catheter often meets no opposition in introduction, because the esophageal muscles are relaxed. The stomach tube removes abundant sour-smelling, sometimes fetid gases and frequently also a rust-brown fluid escapes in a strong stream, particularly after lowering the head.

The circumference of the abdomen is only rarely increased, and even then only moderately; percussion over the abdomen is normal. The intestinal sounds can be heard less frequently, because no food mash gets from the stomach into the intestines as long as the affection lasts; in severe cases the intestinal sounds are suppressed entirely. Cases complicated by catarrh of the intestines or moderate intestinal meteorism are characterized by more intense intestinal sounds. Abdominal pain causes tenesmus and attempts at defecation by reflex irritation. However, since peristalsis is insufficient the animals have a fairly normal defecation only in the beginning of the disease, later on they succeed only occasionally in their attempts at defecation, or constipation becomes complete. Exploration per rectum usually reveals a moderate, exceptionally also an excessive degree of bloating of the small intestines, especially of the duodenum (this part is palpable immediately behind the anterior root of the mesentery, where the duodenum turns over to the left side). The spleen is frequently pushed back, so that its posterior border is displaced towards the region of the external angle of the os ileum. However, it is a mistake to attribute a great diagnostic importance to this change of position of the spleen, as is done by Forssell. For unknown reasons the base of the spleen may become displaced towards the vertical plane of the external angle of the os ileum in health, even in horses which have been starved for days. In small horses one can feel the blind sac of the dilated stomach in front of the left kidney and the base of the spleen either as a tense elastic or as a more or less firm rounded globular body which moves synchronously with the respiratory movements of the diaphragm.

The respiration becomes forced after a short time, a symptom which is markedly in contrast to the almost normal circumference of the abdomen. The pulse rapidly becomes accelerated so that after a few hours the number of pulse beats is over sixty per minute; at the same time its strength becomes diminished; exceptionally the pulse is not more frequent than normally, but it is always weaker. One generally sees an increased filling of the smaller, occasionally also of the larger veins, together with cooling of the peripheral surfaces of the body, cyanosis and perspiration. The temperature usually remains below 39° C.; but in cases complicated with inflammation of the stomach high fever prevails. E. Bauer demonstrated an increase of indican in the urine in primary dilatation of the stomach.

Although the clinical picture of secondary dilatation of the stomach is usually more or less clouded by the symptoms of the underlying primary disease, a diagnosis may possibly be made



on the basis of belching, gagging and vomiting and with the aid of an exploration by means of the catheter. The stomach tube should, therefore, be introduced in all doubtful cases.

**Course and Prognosis.** The symptoms usually subside in mild cases within a few hours and disappear completely after a few more hours; the symptoms of acute catarrh of the stomach, however, not infrequently persist for a few days, and an accelerated and weak pulse may be observable for one or two days. In some cases immediately after the animals appear to have recovered, the symptoms of acute gastric inflammation appears, and they die from it. Pulmonary gangrene develops not uncommonly within a few days, and is due to aspiration after belching or vomiting.

In grave cases (after the observations of the authors in 15 to 33%), the symptoms of gastric dilatation increase in intensity and the patients die with manifestations of great restlessness or after they have already quieted down somewhat. If the patients are treated early with the stomach sound they can usually be saved. Among the causes of death may also be enumerated suffocation, rupture of the stomach, rarely rupture of the diaphragm. The muscularis of the excessively dilated stomach may lose its contractility entirely, so that the pains cease almost completely; but the forced respiration, the very weak pulse and the anxious expression announce the impending suffocation.

After **complete rupture of the stomach** the colicky pains suddenly cease, but the general condition rapidly becomes worse. The animal becomes prostrated, shows extensive muscular tremors, staggers and is bathed in perspiration; in spite of quieting down it does not ingest food (in two cases of Sequens the appetite for food and thirst was retained in spite of rupture); the eyes become staring, the pulse beat rises above 100; but it cannot be felt. Tenderness of the abdominal wall can hardly be demonstrated. If an exploratory puncture is made in the lower part of the abdomen, one can usually obtain a sour or neutral fluid which is hemorrhagic and mixed with particles of feed, particularly grains (Fig. 35). Rectal exploration meets no resistance in the upper portions of the abdominal cavity, because in this part the gases are collected which have escaped from the stomach. In cases which do not take a too rapid course, one can feel particles of food on the peritoneum, which is rough and tender. (Roughness of the peritoneum in itself alone is no proof of rupture of the stomach or peritoneum, because it occurs in peritonitis due to other causes.) The temperature sometimes goes below normal at the start, but rises later if the animals remain alive for any length of time.

Death after rupture may occur within a few quarters of an hour, more rarely only after several hours. If the gastric contents get between the leaves of the mesentery only, the animal may live for several days, and then the symptoms of acute peri-

tonitis can be observed distinctly. Fiebiger saw additional symptoms of emphysema of the skin due to a tear into the parietal layer of the peritoneum.

Pecus claims that rupture of the stomach is announced by a peculiar sharp neighing with subsequent vomiting and the expulsion of the gastric contents through the nose. (It is probable that reference is here made to the peculiar squeaking sound not infrequently heard in horses during a convulsive contraction of the abdominal walls in vomiting; but rupture of the stomach does not necessarily follow the former)

**Rupture of the diaphragm** is announced by the symptoms of collapse and increased difficulty of respiration, also by a tympanic or metallic sound on percussion, which changes from



Fig. 35. *Sediment of the intra-abdominal fluid in rupture of the stomach.* a, large, c, small granules of starch of cereals and leguminose; b, starch granules of oats; d, sarcine; e, white, f, red, blood corpuscles, the size of which varies very much according to the concentration of the gastric contents; g, plant fibers; h, bacilli; i, cocci.

time to time, but is usually found over the left side of the thorax; or, on the contrary, by a circumscribed dullness behind the cardiac region. Exceptionally the diaphragm is pushed forward by the much dilated stomach; the latter together with the diaphragm then comes in contact with the thoracic wall and a tympanic sound is heard behind the cardiac region in spite of the fact that there is no rupture of the diaphragm. Death generally follows soon, but there are exceptional cases; the animal re-



mains alive, a diaphragmatic hernia forms and later on this gives rise to recurrent attacks of disease.

**Diagnosis.** The most important signs for the recognition of acute dilatation of the stomach are the appearance of colicky symptoms after the ingestion of large masses of improper food or after the horse has done work directly after feeding, severe and persisting colicky pains not accompanied by any marked bloating, early appearance of a weak pulse and of difficult respiration, moderate bloating of the small intestines, belching, gagging and vomiting and finally the result of the examination with the stomach tube. Belching, gagging and vomiting or the evacuation of masses of gases and fluid through the stomach tube are also observed in secondary dilatation of the stomach; on the other hand, belching, gagging or vomiting may be absent in primary or secondary dilatation of the stomach. It appears always advisable to examine the abdominal organs by a rectal exploration, because other affections, accompanied by abdominal pains, and similar symptoms, may appear shortly after the ingestion of food. For differential diagnosis are particularly to be considered displacements of the intestines (twisting, volvulus, invagination, internal strangulation), grave forms of thrombosis and embolism and other possible causes of colicky affections.

**Treatment.** The main object of treatment is the evacuation of the contents of the stomach. Mild cases of primary dilatation of the stomach may be treated by laxatives, especially aloes (25.0-35.0 gm.) alone or with neutral salts (100-150 gm.) or rheum (20-25.0 gm.), or salts alone in large doses (sulphate of sodium, sulphate of magnesium) (200-300 gm.). These are best given as pills or electuaries, anyhow with little water. Forssell recommends for the affection 12 gm. of lactic acid (75%) in a pint of water, using it with the apparatus of Goldbech; in dangerous cases a repetition of the dose may be indicated after one-half to three-quarters of an hour. Forssell claims that the administration of lactic acid imparts to the gastric contents that degree of acidity which will favor its removal into the intestines. The experience of the authors, however, teaches that grave cases of gastric dilatation cannot be cured by this treatment and that evacuation by the stomach tube becomes necessary in order to avoid rupture of the stomach or suffocation. One may, on the other hand, inject subcutaneously sulphate of eserine (0.08-0.10 gm.) or arecoline (0.06-0.08 gm.). These drugs have to be used very cautiously, because they may cause sudden death by paralysis of the myocardium or strong contraction of vessels in animals suffering from diseases of the heart or lungs, or they may increase the contraction of the gastric muscularis, increase the pains, cause more reckless rolling and so increase the danger of rupture of the stomach if its contents are not expelled, in spite of their administration.



Chloride of barium should never be used as in other colicky affections of the horse, because occasionally it causes sudden death or a rupture of the stomach. During these years when chloride of barium was used extensively in the Budapest Clinic on horses with colicky affections, fatal results occurred in one third of the cases, due to rupture of the stomach *intra vitam*. Hence this drug is used no longer in horses with colics in the Budapest Clinic and the mortality is less than it was formerly.

Laxatives are of no avail in grave cases, and in these, as indeed in all cases of gastric dilatation, the stomach should be



Fig. 36. Stomach catheter for horses.

emptied with the aid of the stomach tube and should subsequently be irrigated. This is the only rational treatment which can be carried out not only in the clinic, but also in private practice. After the gases and fluids have left the stomach and after the latter has been irrigated, the animals at once become quiet, but the dilatation has been complicated by intestinal ca-

th or intestinal meteorism. As a rule only one evacuation of the stomach is necessary, but occasionally the operation must be repeated.

**Evacuation of the gastric contents by the stomach catheter.** The latter consists of a tube 2.25 meter long, 27 mm. in diameter of ordinary red rubber or the more economical "durit"; its inner diameter is 16 mm. The tube is provided with a flexible rod (cane) of the proper strength and the rod is provided with a conical button at its distal end. (Fig. 36.) This stomach catheter constructed by Marek has the advantage over Monroe's ordinary stomach tube, in that it is less easily damaged by the teeth, and that it will remain in good condition for years if protected against oxidation of fats; however it becomes harder and somewhat thicker if used frequently.

The introduction of this catheter is possible without difficulty in almost any horse with the proper assistance; at least 3 to 4 assistants are necessary; with the animal standing up, introduction is frequently as easy as in cattle. Two or if necessary more assistants draw the head of the animal back and introduce a Bayer mouth-hold between its molars or they insert a mouth gag wrapped in cotton-wool, etc. The assistant who holds the head of the animal with this instrument elevates the lower jaw of the head in a manner to bring it in a line with the neck. The catheter is now drawn laterally out of the mouth, the catheter end to be introduced is made slippery with oil, vaselin or best with glycerine, and is now carefully, but rapidly introduced with both hands along the hard palate into the pharynx and pushed forward until the introduced portion is about to the previously measured distance between the cardiac end of the esophagus and the incisors. After the introduction of the catheter rattling generally occurs, but this is of no significance.

If the catheter meets any resistance before getting into the esophagus, this may be overcome by withdrawing the instrument several times and then pushing it along again or by moving the rod and pouring 1-2 liters of lukewarm water into the tube; this brings about a relaxation of the esophageal wall and of the diaphragm. After the rod has been withdrawn the tube should be well introduced into the stomach, otherwise the procedure may not be successful. (The length of the introduced tube should be 5-125% of the height of the animal, ascertained by a tape measure.)

If there is no evacuation of the stomach after removal of the rod, this may be attempted about by closing the outer end of the tube during inspiration and opening during expiration; in this manner, we make use of the suction produced by negative intrathoracic pressure. In other cases the desired end may be reached by pulling forward and backward on the free end of the tube or by pouring 2-4 quarts of water to run into the tube, while the head of the horse and



Fig. 37. Introduction of the stomach catheter into the horse.



the end of the tube projecting out of the mouth are held down before the water has all run in, producing in this manner syphon-action of the tube. Sometimes a partial removal and reintroduction of the rod may be necessary. The head of the horse must always be lowered considerably if an unimpeded evacuation is to be assured. (The amount of evacuated gastric contents usually varies between 15 and 30 quarts.)

Catheterization of the stomach on the animal in a recumbent posture is almost never necessary and it does not appear to be practicable, since dyspnea may reach a high degree and since the advantage of the syphon action available on a standing animal is lacking.

Catheterization must be performed with care, otherwise fatal injuries to the wall of the esophagus or to the intrathoracic vessels may occur.

The authors cannot recommend the introduction of the stomach catheter through the nasal cavity as proposed by Phillips, because the introduction through the mouth is much easier and permits the use of a much thicker catheter.

**Irrigation of the stomach** becomes necessary in those cases, where the gastric contents are thick-fluid or mashy, or more or less red stained, or when a firm consistency prevents their evacuation. Irrigation of the stomach is carried out in such a manner that the free end of the tube is provided with a funnel through which 2-5 quarts of lukewarm water are allowed to flow into the stomach and are then removed in a manner already described; this procedure is repeated several times. (In a grave case of acute gastric dilatation after the ingestion of corn ears, such irrigation was performed 5 times in 7 hours and a total of 122 quarts of water was used.)

It is necessary in all cases to keep the patients under observation for several hours after the evacuation of the stomach, so that the procedure can be repeated if required by renewed symptoms of colic or by an aggravation of the general condition.

The therapeutic value of the use of the stomach catheter is clearly shown by statistics. The method has for several years been used in the Budapest Clinic; of 85 horses so treated in 1906, 81 (95.3%) recovered. Behrens reported from the Berlin Clinic 93.7% recoveries and other observers reported similarly favorable figures.

Irrigation of the stomach was practiced as early as 1876 by Damman in an experiment foal. A rubber tube closed at one end with a conical plug and provided with openings above was introduced. Grains of oats were removed by this tube, but not particles of hay, evidently because the openings above the closed end of the tube were too small.

The great restlessness accompanying rupture of the stomach may be ameliorated by the injection of morphine (0.3-0.5 gm.) or by the internal administration of chloral hydrate in a mucilaginous medium (Joyeux) or better still as a rectal injection. The intraperitoneal injection of 25-30 gm. of chloral hydrate in ten parts of a 1% sterile salt solution (Breton) speedily quiets the animal; this method of treatment, however, appears dangerous. Rolling and throwing of the animals must be prevented by proper surveillance. Considering the fact that movements hinder the transport of the gastric contents into the intestines, such movements are to be prevented.

After recovery the animals should be starved for one day and they should then receive easily digestible food for some time.

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ness. In carnivora and in rabbits the dilated stomach can be felt through the abdominal walls. Frequently symptoms of bloating of the intestines also supervene. In grave cases the patients succumb within a few quarters of an hour in consequence of rapidly increasing dyspnea, while in milder cases recovery occurs slowly or the clinical picture changes into that of acute gastric catarrh.

The wheezing of fattened hogs is caused, according to Weisz, partially by an acute gastric dilatation after the ingestion of sour bran. A few hours after feeding a forced whistling respiration in a sitting posture is observed, the animals are restless, they groan from time to time and succumb within a short period. (See also page 50.)

In cattle obliteration of the abomasum leads to lack of appetite, also of mastication and secretion of milk, absence of thirst, listlessness, sluggish movements; the animals lie around and will not get up. Harms alone has observed frequent belching and vomiting. The left flank may be sunken in deeply (Harms) or it may be normal or bulging (Koch, Frascch, authors' observation), but it is of normal consistency. The movements of the rumen occur somewhat less frequently, but they become normal after the administration of drugs which stimulate gastric movements, without, however, producing a change in the condition of the animal. A fairly constant symptom is tenderness of the region of the abomasum. Spontaneous groaning is rarely heard. Defecation is at first normal, but becomes less frequent and ceases entirely on the third day of the disease. At this time an increase in the number of pulse beats can be observed and also a moderate elevation of temperature. The disease always ends fatally unless the animals are slaughtered before natural death occurs.

**Diagnosis.** In carnivora, rabbits and hogs the affection can easily be diagnosticated upon the basis of the history and the clinical signs, and in dogs torsion of the stomach can be excluded. Primary obstruction of the abomasum in cattle is suggested by complete lack of appetite from the start, cessation of rumination and of the secretion of milk, with minor disturbances in the functions of the fore-stomachs; the impossibility of influencing the lack of appetite, rumination and defecation by proper drugs; tenderness upon pressure in the region of the abomasum. The exclusion of acute affections of the fore-stomachs is, however, always somewhat difficult.

**Treatment.** The removal of the gastric contents frequently may be brought about by the administration of emetics (apomorphia, veratrine; see page 291). Washing out of the stomach may become necessary; it is easy in carnivora, and the principles of the procedure are the same as in the horse; the stomach

tube which is used must, of course, be much thinner. In obstruction of the abomasum in cattle, treatment with laxatives (see page 254) has never been successful.

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## 6. Chronic Dilatation of the Stomach. *Dilatatio ventriculi chronica.*

Chronic dilatation of the stomach is an enlargement of the stomach which persists, at least for some time, even after the removal of the cause or after evacuation of the gastric contents, and which may be likened to chronic atony of the fore-stomachs of ruminants.

**Etiology.** Persistent dilatation of the stomach frequently occurs after feeding, for a long time, non-nutritious feed, such as chaff, coarse straw, coarse leguminosæ. Such food is also detrimental because the animals are forced to ingest larger amounts in order to satisfy their requirements. Animals which are excessive feeders are in particular danger. The gastric muscularis is too weak in some individuals to propel a feed mash of this kind, consequently the food remains too long in the stomach; it becomes decomposed and in the course of time causes a chronic dilatation of the stomach. Dilatation of the stomach on this basis has been observed by Trinchera in 2-3% of young horses, mostly three to five years old, which had been imported from the provinces of Carinthia, Salzburg and the Tyrol to Italy, and it has also been observed in horses from the north of France, from Croatia and Dalmatia. Sand and gravel swallowed in large amounts exert a similar effect. (Dilatation of the stomach due to an accumulation of sand combined with an accumulation of sand in the intestines frequently occurs, according to Wedernikow, in the Steppes of Kirgisia (Siberia) and is known as Gata-disease (see page 292).

Persistent obstruction at the pylorus (pyloric stenosis due to tumor formation or to cicatricial contraction) or in the small intestines (stenosis, frequent obstipation, intestinal calculi) usually leads to chronic dilatation of the stomach. The cause of this affection may also be found in atony of the gastric muscularis due to chronic catarrh of the stomach, to tumors of the stomach, to sclerosis of the muscularis (Liénaux), or to chronic interstitial hepatitis of horses (socalled Schweinsberger disease); in the last named disease the ingestion of food which is difficult of digestion plays an important rôle. Acute dilatation of the stomach, occurring a number of times, may likewise be the cause of chronic dilatation (especially in dogs) and continued swallowing of air may also lead to this chronic affection.

Since the muscularis of the stomach becomes weakened with advancing age and since mastication likewise becomes deficient, chronic gastric dilatation is frequently seen in older animals.

**Pathogenesis.** Most of the causes enumerated first produce hypertrophy of the gastric muscularis which enables the stomach to furnish the required increased amount of work. After the ingestion of food, particularly in horses, the stomach is stimulated to stronger even to convulsive contractions, which have the object to propel the gastric contents into the intestines. It is possible that the stomach gets rid of its contents in this manner, though after a considerable time. By and by the muscular power decreases, the food remains longer in the stomach, it ferments and the stomach becomes more and more dilated. According to its size the dilated stomach forms an impediment to respiration and influences the blood circulation unfavorably, especially when it is filled with food stuffs. The disturbances are most marked right after the ingestion of food and they may amount to pain. As the dilatation increases, the muscularis of the stomach finally loses its contractility completely and the gastric contents cannot be removed any more.

**Anatomical Changes.** The stomach is sometimes increased to several times its normal size, and in grave cases its shape may also be changed (the stomach of the horse becomes egg-shaped or similar in shape to the rumen of cattle), its wall is generally thickened, occasionally also thinned, sometimes as thin as brown paper (Fitzroy-Philipot), sometimes also fairly tough (Liénaux). The mucosa generally presents the signs of chronic catarrh. Sometimes we find as the cause of a dilatation a tumor in the stomach, in the pylorus or in the intestines.

In a case of Koch, the stomach of a horse weighed 51 kilograms when full and had a cubic content of 84 liters (the normal cubic contents being 7 to 15 liters); its largest diameter was 164 cm. Fitzroy-Philipot found a full stomach of a horse weighing 71 kg., it weighed 10 kg. (about 20 pounds) when empty and its length (between cardia and pylorus) was 261 cm., the greatest thickness 190 cm. Liénaux found the larger curvature in an adult setter dog to be 50 cm. long. However, increases of this extreme degree are seen only exceptionally.

**Symptoms.** Horses suffer in this affection frequently from repeated attacks of colic (so-called chronic or periodic colic) which are much like those seen in acute dilatation (see page 299). Such attacks occur in stenosis of the pylorus almost regularly towards the end of feeding or immediately after it; in other cases shortly after the meal; they frequently last for several hours. Each attack may, of course, become fatal in consequence of suffocation, rupture of the stomach, etc. In the further course of the disease the attacks of pain become less severe; however, the general condition does not improve and the attacks last a longer time; the animals suffer more and more in their general condition. Respiration is also difficult in the periods



between the attacks, and some animals may develop roaring. Finally, when the gastric muscularis has lost its contractility, no more attacks of pain occur, but the patients soon succumb to exhaustion, rupture of the stomach, suffocation or peritonitis.

In other cases, when dilatation is due to catarrh of the stomach, or to improper food, one sees from the start the clinical picture of catarrh of the stomach in combination with difficult respiration, if the dilatation is at all considerable. In such cases and in those described above, the dilated stomach may be palpable from the rectum. During the slow development of the dilatation the organism has time to adapt itself to changed conditions, hence during rest the pulse does not present a marked deviation from the normal. However, rupture of the stomach may occur at any time.

Trinchera has described a condition under the name of "abdominal foundering of young horses," which is due to gastric dilatation after improper feeding, and which is characterized by slow and hesitating inspiration, followed by quick interrupted expiration with drawing in of the flanks and bulging of the region of the lower ribs. These disturbances are most marked during and after the ingestion of food. If proper feeding is instituted the disturbances disappear after an interval which is longer than that during which the affection has persisted.

These observations of Trinchera are in accord with others made in the province of Salzburg and showing that the permanent ingestion of voluminous food or the sojourn in poor pastures is followed by similar disturbances in respiration which however disappear on proper feeding (change of owner).

Oppenheim observed a case of chronic dilatation of the abomasum in cattle. The animal vomited daily for weeks, especially at the beginning of rumination. In a case described by Koch the affection followed upon atony of the fore-stomachs and its symptoms were obscured by those of the atonic condition of the latter.

In dogs the symptoms are those of chronic gastric catarrh. The filled stomach may be palpated in both regions of the lower ribs; if its space is not completely filled with masses of food, percussion shows a tympanitic sound over a space between the eighth rib and the umbilicus (Schindelka); this sound becomes weaker or disappears after the ingestion of food; sudden pressure upon the gastric region elicits splashing sounds.

Liénaux observed dilatation of the stomach of a dog, which appeared simultaneously with a sclerosis of the muscularis. The affection at first came on in periodic attacks, it then became permanent and led to diarrhea, which resisted every treatment, the feces contained almost unchanged food, particularly meat. The appetite was good. Behind the diaphragm one could feel a large immobile mass. The absence of vomiting was believed to be due to the stiffness of the stomach wall.

**Treatment.** If treatment is at all desirable, the ingestion of easily digestible, or still better, fluid feed (see page 291) may retard the development of fatal symptoms and the general condition of the animals may be preserved to a certain degree.

To remove the accumulated gastric contents the same means may be employed as recommended for gastric atony or for acute dilatation of the stomach (see pages 273 and 303). Pyloric stenosis in dogs may be treated by a gastroenterostomy as practiced in human surgery; this operation has often been performed successfully in experiments on dogs.

**Literature.** Castel, *Rev. vét.*, 1903, 472 (Lit.).—Fitzroy-Philipot, *J. vét.*, 1885, 108.—Hitze, *Pr. Mil. Vb.*, 1905, 161.—Koch, *Pr. Mt.*, 1880-81; 58, *B. t. W.*, 1890, 195.—Liénaux, *Ann.*, 1898, 65.—Oppenheim, *T. Z.*, 1899, 139.—Trinchera *Clin. Vet.*, 1907, 613.

## 7. Foreign Bodies in the Stomach. *Corpora aliena ventriculi.*

**Etiology.** Aside from what occurs in rabies, in playing, retrieving or also in devouring their food, dogs frequently swallow foreign bodies, such as stones, pieces of money, corks, bits of glass, skeins of twine, pieces of cartilage, bone, rags, sponges, needles, etc.; hair balls are rarely found in the stomach of dogs.

In cats, pieces of mice or rats, or whole mice, are sometimes arrested in the stomach, other foreign bodies only very rarely; such foreign bodies may be needles, fruit seeds, etc.

**Pathogenesis.** As long as foreign bodies remain free in the stomach, they usually do not produce any harm, but according to their nature, they frequently lead to acute or chronic catarrh or inflammation of the stomach. Sharp or pointed bodies frequently injure the gastric mucosa and penetrate it, leading then to perforation or fistula. Pointed as well as dull bodies may become wedged in the pylorus and may bring about its complete obstruction.

Swallowed pins rarely cause gastro-intestinal disturbances. Zoja, who experimented on cats, made them swallow 125 sewing pins; only two penetrated, one above the pylorus, one into the rectum; the others were either voided through the anus or were found in the killed animals in the large intestine where they had not led to any injury (Eichhorst).

**Symptoms.** These occasionally are similar to those of an acute, more rarely of a chronic gastric catarrh; they are more frequently similar to those of inflammation of the stomach; the lodgment of the foreign body in the pylorus produces the symptoms of obstruction of the pylorus. In the latter case the following symptoms are observed: Obstinate vomiting, lack of appetite, sometimes intense thirst, excitement, frequent change of posture, loud howling, groaning, inclination to bite, convul-

sions, finally complete listlessness. The stomach is tender to pressure and the foreign body may occasionally be felt in the region of the lower ribs behind the liver (Udriski felt the end of a hairpin in a cat). The weakness rapidly increases, fever sets in, the pulse becomes rapid and weak and the animal succumbs.

**Diagnosis.** On the basis of the symptoms described and in connection with the history of the case a diagnosis can, as a rule, be secured. Occasionally one might resort to an exploration of the abdominal cavity by the aid of Roentgen rays. However, it must not be forgotten that a large portion of the stomach in a Roentgenogram is covered by the liver, hence bodies which have not a high specific gravity may not become visible. It is advisable when making an X Ray plate to have the animals on their hind legs, so as to bring the pylorus below the liver. The stomach might also first be filled with carbon-dioxide gas.

**Treatment.** The removal of a foreign body which is neither sharp nor pointed may be attempted by an appropriate emetic (apomorphine 0.005-0.01 gm.). If this treatment is not successful, or if it cannot be tried on account of the nature of the foreign body, laparogastrotomy is indicated. With its aid Hamoir, Vennerholm, Udriski and Bergeon have saved dogs and one cat.

**Literature.** Bergeon, *Rev. vét.*, 1905, 698.—Eichhorst, *Physik. Untersuchungsmethod.*, 1881, 241.—Hamoir, *Ann.*, 1897, 649.—Mueller, *D. t. W.*, 1907, 570.—Udriski, *Arh. vet.*, 1904, 160.—Vennerholm, *Z. t. Tm.*, 1897, I, 123. (See also *Lit. of intestinal obstruction in carnivora*.)

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**Foreign Bodies in the Stomach of Other Animals.** Foreign bodies are only very exceptionally found in the stomach of the horse. Hahn and Marek each found a piece of wood, Eberbach and Enke needles, respectively, 21 and 6 cm. long, Wölker and Grundmann found pointed fragments of bone in the stomach or its neighborhood in the cadavers of horses which came to post-mortem examination. Foreign bodies cause inflammation of the stomach, perforation of this organ, or inflammation of the neighboring organs and of the peritoneum. Schmidt saw traumatic inflammation of the stomach, diaphragm and lungs of a horse. In a few cases feed-balls (Bayer) or gastroliths were found on post-mortem examination in horses which had died with symptoms of colic (in a case of Moebius the stone found in the stomach weighed 324 gm.; in a case of Johnes 750 gm.).

In the stomach of swine foreign bodies occur somewhat more frequently (pointed objects, pieces of wood, hair balls), with the exception of hair balls, these usually lead to perforation of the stomach and to secondary peritonitis.

In fowl the opening of the glandular or muscular stomach is very exceptionally obstructed by foreign bodies (see plate III, page 244); this generally causes death after lack of appetite and emaciation.



domestic animals, as it does in man is a point not yet settled in veterinary science.

One must also admit the possibility that colonies of multiplying bacteria may cause necrosis and ulceration in the gastric mucosa.

**Anatomical Changes.** Ulcers are generally found in the neighborhood of the pylorus; they are round, oval or irregularly shaped and their base is either formed by the denuded but otherwise unchanged muscularis or serosa or by pseudomembranes covering the peritoneal coat. The margins of the ulcers, particularly those of peptic ulcers, are quite sharp as if they had been stamped out artificially. Peptic ulcers (*ulcus pepticum sive rotundum, sive corrosivum, sive e digestionem*) do not show an inflammatory reaction in their neighborhood, they imitate in their outlines the shape of a funnel placed with its long axis obliquely towards the pylorus. If an ulcer perforates, general peritonitis occurs unless adhesions have previously been formed with the neighboring organs. Erosion of a larger vessel leads to copious hemorrhage.

**Symptoms.** Gastric ulcers have generally been found in domestic animals accidentally upon post-mortem examination (in slaughtering horses without preceding observation of characteristic symptoms during life). If there are any observable symptoms during life they are identical with those of chronic gastric catarrh (see page 293) or chronic atony of the proventriculi (see page 268). One may be justified in thinking of gastric ulcer if there is occasional hematemesis (vomiting of blood) or if the feces are hemorrhagic, black, tarlike. In horses mild colicky attacks have occasionally been observed. The condition of the patients often improves remarkably within a few days and then there is suddenly a marked relapse.

Gotteswinter observed a case in a cow where profuse hemorrhage led to profound anemia. A similar observation was made by Kitt: Kohlhepp saw in a cow the formation of an extensive emphysema of the skin, which created the suspicion of an attack of black leg (however, it was simply a case where gases from the stomach had found their way through an ulcer into the subcutaneous tissues). In a case of Plate a tuberculous ulcer in the abomasum caused a fatal hemorrhage

Ulcers may exist for a long time in the stomach, so that adhesions to neighboring organs may be formed, which will then prevent perforation. If the latter occurs anyhow, peritonitis sets in and rapidly takes a fatal course. According to Rasmussen one must think of round ulcer if calves become restless during transportation, throw themselves down, and then lie apathetically on the floor.

**Treatment.** When there is suspicion of ulcer of the stomach, undigestible, coarse feed should be avoided because it in-

creases the danger of perforation. Mucilaginous and astringent drugs are indicated (subnitrate of bismuth, silver nitrate), also hemostatics including adrenalin or suprarenin (for dogs 30 drops of the 1% solution internally, repeated several times). The administration of hydrochloric acid is contraindicated. Carlsbad salt in larger doses may be employed with advantage, since it neutralizes an excess in hydrochloric acid, diminishes gastric secretion and aids in the removal of the contents of the stomach. Obstinate vomiting must be met by narcotics (opium).

**Literature.** Eisenmann, *Monh.*, 1906, XVII, 97.—Groll, *W. f. Tk.*, 1907, 627.—Jagnow, *Z. f. Tk.*, 1900, 172.—Joest, *Dresd. Ber.*, 1908, 140.—Kitt, *Munch. Jhb.*, 1879-80, 31; 1892-93, 28; *Monh.*, 1899, X, 28.—Kohlhepp, *B. Mt.*, 1886, 197.—Kolb, *D. Z. f. Tm.*, 1890, XVI, 291.—Moussu, *Bull.*, 1895, 100.—*Munch. W. f. Tk.*, 1876, 385.—Nagel, *D. Z. f. Tm.*, 1889, XV, 128.—Plate, *B. t. W.*, 1906, 713.—Rasmussen, *D. Z. f. Tm.*, 1891, XVII, 458.—Roloff, *Pr. Mt.*, 1867-68, 157.—Steinhardt, *Z. f. Vk.*, 1908, 313.—Zippelius, *W. f. Tk.*, 1875, 357.

### 9. Neoplasms in the Stomach. Tumores ventriculi.

**Occurrence.** Tumors in the wall of the stomach are very rare, and therefore, and because they are difficult to diagnosticate, they do not have a great deal of practical significance.

In the **fore-stomachs of ruminants** the omasum appears to be the favorite seat of neoplasms; here there are found polypoid tumors which attain a size of two fists and which may close up the opening between reticulum and omasum. Similar tumors are found more rarely in the reticulum. Sarcomata are likewise found in the omasum, up to the size of a fist; nodular, tough tumors, generally showing necrotic changes; these tumors are found more rarely in the reticulum or rumen. Kitt saw a chondroma in the wall of the rumen of a calf. Finally the rumen of cattle may contain ulcers as large as a hand, the base being formed by thickened nodular masses of the wall of the rumen; such ulcers are probably due to carcinomatous neoplasms.

Tumors are common in the **stomach proper** (the abomasum), where they are found in the pylorus, the cardia, and the larger curvature. In the horse there occur sarcomata and carcinomata which, when growing in the stomach, may obstruct the cardia, the pylorus and the duodenum. In the abomasum of cattle, epithelial neoplasms are met with, such as adenomata, adeno-carcinomata, alveolar carcinomata, which undergo an early disintegration; further, diffuse lymphadenoid infiltration. Joest found a tuberculoma in the stomach of a cow with general tuberculosis. Eberlein observed a primary carcinoma in the stomach of a dog. More rarely are lipoma, fibroma, myoma, found in the stomach; similar to the latter is the circumscribed muscular hypertrophy which is sometimes met with in the stomach of the horse.

**Symptoms.** The presence of a tumor of any size in the stomach disturbs its function on account of the destruction of

part of the mucosa and on account of the impediment to the proper movements of the organ. Disturbances of digestion are, therefore, observed, which, however, have nothing characteristic, but are identical with those seen in chronic gastric catarrh or in atony of the fore-stomachs. In horses symptoms of colic are also seen occasionally after the ingestion of food; further, dizziness and vomiting; in cattle chronic bloating, frequent belching, vomiting; in dogs enlargement of the circumference of the abdomen, vomiting, icterus. The animals become more and more emaciated, cachexia sets in and they finally die after complete exhaustion. Exceptionally a disintegrating neoplasm may lead to perforation of the stomach wall or to internal hemorrhage (Menges) in consequence of the erosion of a larger blood vessel.

**Diagnosis.** It is very difficult to diagnosticate a tumor of the stomach; in large animals it is almost impossible to be at all certain, but in carnivora tumors of the stomach, particularly in the region of the esophagus, may sometimes be mapped out by palpation. In a case of Hartmann the clinical picture of a carcinoma of the stomach was very much like that of stenosis of the esophagus.

**Treatment.** Only in carnivora is there any hope of recovery if the operative removal of the tumor can be carried out successfully. Parascandolo performed gastrectomy in a dog with good success.

**Literature.** Ball, J. vét., 1906, 709.—Cadéac, Rev. vét., 1885, 434.—Darmagnac, Rev. gén., 1905, V, 609.—Eberlein, Monh., 1897, VII, 289.—Hartmann, Z. f. Vk., 1905, 157.—Joest, Dresd. Ber., 1907, 169.—Menges, Vet. Jhb., 1885, 87.—Messner, D. t. W., 1909, 19.—Parascandolo, W. f. Tk., 1901, 45.—Petit & Fayet, Bull., 1902, 648.—Savary, Rev. vét., 1903, 177.—Utz, B. Mt., 1889, 110.—Zietschmann, S. B., 1908, 73.

## 10. Torsion of the Stomach. Torsio ventriculi.

(*Volvulus ventriculi*.)

In dogs a change of position of the stomach is observed occasionally, so that the organ with the pylorus turns around the cardia from right to left, consequently the larger curvature is to the right and the pylorus towards the region of the left lower ribs. Exceptionally there occurs torsion of the abomasum in cattle.

**Occurrence.** From observations made, it appears that torsion of the stomach in dogs is not a very rare occurrence. Kitt was the first to describe two cases, later on the disease has been observed more frequently by Cadéac in Lyons, Jensen in Copenhagen, Johné in Dresden. Single cases have also been reported by Wallmann, Poeanaru & Slavu, Rehabe, Billingham and



Ellermann; Bonvinci reported one case in Italy, v. Ratz two cases in Hungary, Markus five cases in Holland.

Only one case of torsion of the rennet in a calf eight days old has been reported by Carougeau & Prestat.

**Etiology.** It is owing to the great mobility of the pyloric half of the stomach, including the pylorus in dogs, that in certain movements, such as jumping, rolling, rapid running upstairs, the stomach easily changes its position. Since the stomach is sufficiently fixed by the abdominal wall and the abdominal organs, when completely filled, it can only change its position either if it is entirely empty or if it contains such bodies as pieces of bone, meat or liver which may produce a swinging motion of the stomach during the indicated movements.

**Pathogenesis.** The pyloric half of the stomach and the duodenum get from behind the surface of the liver on the right side of the abdominal cavity, into the left side between the abdominal portion of the esophagus, the cardia of the stomach and the liver, where it will be compressed; at the same time the abdominal portion of the esophagus turns in a direction like the hand of a clock (seen from behind). In consequence of this change of position, both openings of the stomach become closed, the veins of the stomach and those of the spleen, which is forced to follow the change of position of the stomach, become partially or completely obstructed in consequence of pressure and torsion. The less collapsible and more resistant arteries, in which there is a high blood pressure, continue to propel an almost normal amount of blood into the vessels of the stomach and spleen, and a rapidly increasing profound venous congestion is developed. Excess of carbon-dioxide in the gastric vessels causes convulsive colicky pains and contractions of the gastric wall, which will only cease after several hours and after bloating and edematous infiltration of the gastric wall have developed. Pain is also caused by a torsion of the nerves and by the development of peritonitis. Since nothing can pass from the stomach and since absorption into the blood is almost abolished, the stomach becomes quickly and enormously dilated by gases which develop from food stuffs that may be present, and from the hemorrhagic infarction with serum extravasation, which has been caused by the torsion. The more feed there is present in the stomach, and the more severe the twisting, the more rapidly will bloating attain a dangerous degree. The enlarging stomach interferes with the movements of the diaphragm and in this manner and also in consequence of a developing general infection, eventually also of peritonitis, which is caused by microbic invasion through the damaged stomach wall, disturbances of circulation are produced.

**Anatomical Changes.** Sometimes the stomach is enlarged and bloated to ten times its normal volume; the pyloric half and

the first portion of the duodenum are found between the posterior surface of the liver and the posterior surface of the stomach, which are now directed towards the head below or, on the contrary, above the twisted esophagus, while the great curvature is turned towards the right. The mesentery of the duodenum forms a tense string drawn between the right kidney and the stomach and compressing the median part of the latter from in front. The mesentery is drawn over the dilated stomach and the dark red wall of the latter may be seen shining through it. Upon cutting open the stomach, gas is discharged abundantly; the mucosa is dark red; the whole wall is infiltrated and edematous. The dark red discoloration is sharply demarcated towards the esophagus, but fades out gradually at the pylorus towards the normal mucosa of the duodenum. The spleen is much enlarged and curved in the shape of a capital U, blackish red in color, the capsule is tense. The abdominal cavity usually contains a hemorrhagic serous fluid. The liver is somewhat displaced towards the right and is anemic. Occasionally there may be signs of a beginning peritonitis.

In the case reported by Carougeau & Prestat, the abomasum of the calf was twisted towards the right; both openings were closed; its wall showed evidences of much congestion, the peritoneum was inflamed.

**Symptoms.** The disease is characterized by a sudden attack of abdominal pain, and by rapidly increasing bloating. The patient stands motionless and apathetic on one spot and if urged on, moves stiffly and carefully. The ingestion of food and drink, and defecation cease; exceptionally there may be intense thirst (Bonvicini), but the ingested water at once returns through the mouth or nose. Gagging sometimes supervenes, but vomiting does not occur; irritation of the fauces causes retching, but no vomiting. The abdomen usually rapidly enlarges in all directions, in exceptional cases only slowly (Bonvicini), it appears tense. The percussion sound is everywhere loud, occasionally of a metallic character with the exception of a dullness in the right hypochondriac region as large as a hand, which is caused by the spleen which is here situated behind the abdominal wall (Cadéac). Palpation of the abdomen causes intense pain, particularly after the affection has lasted a certain length of time; one can feel a tense sac in the abdominal cavity. Intestinal sounds are absent.

As the abdomen gradually increases in size, difficulties in respiration increase to attacks of suffocation; cyanosis becomes marked, the heart beat is much accelerated, hard; the pulse becomes gradually weaker and finally it can no longer be felt.

The disease is of short duration, death ensues within 24 to 48 hours after the appearance of the first symptoms, as a rule in consequence of suffocation.

In the case of Carougeau & Prestat, the previously healthy calf stopped sucking, protruded its tongue, did not lie down, defecated less frequently, colicky pains and constipation soon came on. After the administration of milk the restlessness increased. The next day weakness was already well marked and pressure upon the abdomen elicited splashing sounds. After a day and a half the calf died.

**Diagnosis.** The clinical picture in the dog is characteristic enough to enable us to make a diagnosis; an exploratory puncture may however become advisable. In obstruction of the esophagus there are no abdominal pains nor bloating. In displacements of the intestines there occurs true vomiting, bloating of the abdomen is absent or quite insignificant. In the course of general acute peritonitis vomiting is frequent, fluids poured into the mouth get into the stomach, bloating is not very marked and there is fever from the start.

**Treatment.** In cases which are not yet far advanced operative interference may bring relief. To diminish the tension of the abdomen and to relieve the lungs it is necessary slowly to remove the gases from the stomach by puncture. Then the abdominal cavity is opened and the collapsed stomach replaced in its original position. This is done with the fingers introduced between the stomach and liver, which then grasp the pylorus and the duodenum and twist them back to their normal place. (In a case not completely elucidated Cadéac brought about recovery by a laparotomy.)

**Literature.** Bonvicini, *Nuov. Erc.*, 1900, 290.—Cadéac, *J. vét.*, 1895, 513, 1906, 16.—Carougeau & Prestat, *J. vét.*, 1898, 340.—Jensen, *Maanedsskr.*, 1899, X, 70.—Johne, *S. B.*, 1902, 217.—Kitt, *Monh.*, 1895, VI, 20.—Poeanaru & Slavu, *Arh. vet.*, 1906, 185.—Behaber, *W. f. Tk.*, 1907, 681.—Wallmann, *B. t. W.*, 1897, 38.

**Other Changes of Position of the Stomach.** In dogs, the stomach sometimes slips through an opening in the diaphragm into the thoracic cavity (Fünfstück, Johne, authors' observation); a case of Baerner shows that the horse's stomach may also exceptionally get into the thoracic cavity. In ruminants, the reticulum and occasionally the omásium may enter the thoracic cavity through an opening of the diaphragm. A prolapse of the stomach in a hog, which appeared to be healthy during life, was found by Fritz.

An auto-invagination of the stomach was reported by Adam, invagination of the stomach into the duodenum by Spencer.

If strangulation of the prolapsed stomach takes place, the animals which have already suffered from difficult respiration succumb in a short time to suffocation. In the case of Baerner a roaring horse with a partially prolapsed non-incarcerated stomach had occasional attacks of colic. In ruminants, prolapse of the fore-stomachs causes chronic bloating, possibly also compression of the heart.

**Literature.** Adam, *W. f. Tk.*, 1866, 63.—Bärner, *Z. f. Thl.*, 1899, III, 333.—Fritz, *Schw.*, A. 1904, XLVI, 106, 164.—Fünfstück, *S. B.*, 1878, 106.—Spencer, *J. of comp. Path.*, 1890, 45.



**11. Hemorrhage from the Stomach. *Hæmorrhagia ventriculi.***

(*Blutbrechen*, [German]; *Hæmatemesis*.)

**Etiology.** Arterial hyperemia of the gastric mucosa easily causes hemorrhages per diapedesin or per rhexin, which are rarely so abundant that there is a free blood extravasation into the cavity of the stomach. This may however be the case if acrid or caustic substances have caused a deeply penetrating inflammation or a necrosis of superficial layers of the mucosa with subsequent ulceration.

Hemorrhages from the stomach likewise appear in the course of certain acute infectious diseases; they may then be due to local inflammatory processes or more generally to the general infection. Diseases of this type are particularly purpura hæmorrhagica, anthrax, hog cholera, swine plague, distemper in dogs, smallpox, scorbutus, etc.

Diseases of the blood vessels and changes in the blood are the cause of hæmatemesis which occurs in blood diseases and cachexia. Ulcer of the stomach gives rise to hemorrhages from the stomach. Swallowed foreign bodies sometimes injure vessels of the gastric wall and cause hemorrhages into the stomach, gastric vessels may also be torn in a fall or in overwork. In horses gastrophilus larvæ sometimes produce injuries which lead to hæmatemesis (Qualitz). Rupture of the enlarged and subsequently adherent abdominal aorta or of the celiac artery may also form an extremely rare cause of fatal hemorrhage into the cavity of the stomach.

Congestion of the gastric mucosa leads usually at most to parenchymatous hemorrhages, as is seen in the course of diseases of the liver, of the portal vein, of the heart and of the lungs.

**Symptoms.** The only reliable symptom of hemorrhage of the stomach consists in the vomiting of blood (*hæmatemesis*, sive *vomitus cruentus*); it is however usually observed only in carnivora and then only after more severe hemorrhages. The animals then expel either feed-material streaked with blood or pure blood through the mouth and nose. The latter may be fluid and bright red or coagulated into dark brown masses. After having been in the stomach for a longer time, the vomited blood is dirty brown or of a chocolate color, similar to ground coffee, since the hemoglobin has been changed into hematin by the action of the hydrochloric acid of the gastric juice. Microscopic examination of the vomited masses which have an acid reaction or of the gastric contents removed by catheter shows, aside from more or less changed particles of food, pale or disintegrating blood corpuscles, although they may be entirely absent, having been completely dissolved by the gastric juice.

Most frequently blood which has been extravasated into the cavity of the stomach enters the intestinal tract and leaves the body with the feces. During its way out it is always changed materially; the hemoglobin is decomposed and the albumen undergoes putrefaction, hence the feces are tar-like discolored, soft, or on the contrary dry and very fetid.

If the hemorrhage is profound the general symptoms of internal hemorrhage become manifest; pallor of the mucous membranes, small pulse, perspiration, dizziness; repeated small hemorrhages lead to chronic anemia.

**Diagnosis.** Gastric hemorrhage can be diagnosticated only upon the basis of hematemesis, or upon the removal of blood-tinged masses with the stomach tube and then only if it can be ascertained that the blood comes from the vessels of the stomach and did not get into the latter from some other place. The difference between pulmonary hemorrhage and hematemesis has been pointed out elsewhere (see page 89); it should here again be mentioned that blood may get into the stomach from the nose, from the buccal cavity and from the respiratory tract; such hemorrhages must be excluded before a diagnosis of gastric hemorrhage can be made reliably. There is also the possibility that dogs may vomit previously ingested blood; this is often indicated by the fact that no anemia can be observed in spite of large masses of blood vomited. Whether blood voided with the feces comes from the stomach or from the anterior portion of the intestinal tract cannot be determined solely by the appearance of the feces; the clinical history of the case and the examination of all other organs must determine the diagnosis.

**Treatment.** In order to stop the hemorrhage the patients should be placed at rest and should be made to swallow ice or some cold water; in dogs external applications of ice may be made in the region of the stomach. Subcutaneous injections of ergotin (5-10.0 gm. or 0.2-1.0 for small animals), extr. hydrastis (8-10.0 gm. or 0.2-0.5 gm.) are used with advantage. To diminish the movements of the stomach opium internally (5-10.0 or 0.1-0.5 gm.) is useful, eventually combined with astringents (acetate of lead, tannin, tannoform, tannalbin, sulphate of iron, chloride of iron, alum). Gelatin may also be used either alone or with wine internally; also adrenalin or suprarenin (for dogs every hour 30 drop doses of the 1% solution). Caution is necessary in internal medication since it may further stimulate vomiting.

As long as the hemorrhage lasts, the animals should be starved, then they should receive only fluid, soft and mashy feed for several days.

**Literature.** Qualitz, Z. f. Tk., 1891, 459.—Schneidemühl, Tm. R., 1888-89, III, 13.—Vogel, Rep., 1863, 116.

## 12. Intestinal Hemorrhage. Enterorrhagia.

**Etiology.** More severe hemorrhages upon an inflammatory basis are usually observed only if the inflammation has been caused by an infection or by acrid caustic materials or if it is complicated by ulceration. This is seen in the course of anthrax, cattle plague, hog-cholera, swine plague, distemper in dogs, pyemia, septicemia; after intoxication by caustic poisons, in meat poisoning, after the administration of drastic laxatives. Intestinal hemorrhages are frequently seen during purpura hemorrhagica, scorbutus, and other diseases, and are due more to morbid changes of the vessel walls than to a local disease of the mucosa.

Mechanical injury of the intestinal mucosa by swallowed foreign bodies (fragments of bone, nails, etc.), animal parasites, echinorrhynchus gigas, taenia echinococcus, dochmius duodenalis, etc., desiccated coproliths, hard substances introduced into the rectum (end-piece of a rectal syringe), or stabs produced intentionally (Sequens) may cause greater or lesser intestinal hemorrhages.

Congestion of the intestinal veins in chronic interstitial hepatitis or in valvular disease of the heart, obstruction or compression of the portal vein, thrombosis or embolism of intestinal arteries may occasionally be the cause of intestinal hemorrhages. Hemorrhoids (dilatation of veins) below the rectal mucosa, which is sometimes seen in dogs and horses (Utz, Eckart) also are the cause of hemorrhages from the rectum.

As further causes of intestinal hemorrhages there may be mentioned: very vascular or disintegrating tumors, especially mucus polyps, intussusception, tearing of the dilated abdominal aorta or of the anterior mesenteric artery after they had previously become adherent to the intestinal wall, finally extensive burns, probably on account of the formation of emboli in the intestinal vessels.

**Symptoms.** The feces are stained with blood, or pure blood may occasionally be voided through the anus. The more profuse the hemorrhage, the more easily blood coagula are formed, so that the voided masses may sometimes consist of coagula only. This is particularly observed in hemorrhage from the posterior portions of the intestines, since blood here does not mix well with the already firm feces. In hemorrhage in the anterior portions of the intestines, the feces are intimately mixed with blood, they are tar-like, black, or even as though charred, and they have an intense putrefactive smell. If the hemorrhages have been only slight the feces appear pale reddish or only streaked with blood; in slight rectal hemorrhages the bloody streaks are limited to the surface. Defecation may be very easy or it may be quite difficult, particularly if the hemorrhage is due to inflammatory conditions.



Microscopic examination may show the presence of blood even if it cannot be discovered with the naked eye. The red blood corpuscles may be intact, or they may be swollen, pale, broken up, or they may be entirely dissolved if the hemorrhage has occurred in the anterior portions of the intestines; then positive tests for blood-pigment alone can demonstrate that a hemorrhage had taken place.

Aside from local symptoms, the symptoms of anemia present themselves in proportion to the amount of blood lost. (See Vol. I.)

In a horse with two hemorrhoidal nodules of the size of a walnut in the mucosa of the rectum, Utz observed recurrent attacks of colic, with the discharge of fluid blood or dark coagula. The horse finally succumbed to anemia and hydremia.

**Diagnosis.** As a rule blood can be recognized in the feces with the naked eye. One may however be misled by a naturally dark brown color of the dog feces; or by the brown color of feces of animals which have received preparations of bismuth or iron. In doubtful cases, it is necessary to resort to microscopic, chemical and spectroscopic examinations, and to the test of mixing the feces with water to see whether the latter is stained red.

The site of a hemorrhage is indicated to some extent in so far as the blood is changed less, the shorter the distance it has traveled towards the anus; the presence of streaks of blood on the surface of otherwise normal feces indicates rectal hemorrhage, while an intimate mixture of the blood with the feces points to hemorrhage in the small intestines. However hemorrhage from the stomach may give rise to bloody feces and the blood may even have reached the stomach from parts still nearer to the head. It is also necessary to remember that dogs (particularly butchers' dogs) may have ingested the blood of slaughtered animals or raw meat and then their feces may also contain blood pigment; the general condition of such animals is of course normal.

The exact determination of the site of a hemorrhage often meets with great difficulties and necessitates a careful examination of all of the organs and a consideration of the whole history of the patient. The feces are not always bloody in all cases of intestinal hemorrhage, because the animal may either die before the blood reaches the rectum, or mechanical obstructions may interfere with its passage to the rectum; these conditions are frequently prevalent in thrombosis of the mesenteric vessels and in hemorrhages due to intestinal invagination, in torsion (volvulus) or in strangulation.

**Treatment.** The treatment of intestinal hemorrhage is in general identical with that of hemorrhage from the stomach, and preparations of opium and astringents may be administered every one to two hours without any fear. Hemorrhages from the posterior portion of the intestinal tract require local treat-

ment from the rectum with gelatin dissolved in hot water, astringents, also adrenalin or suprarenin (see page 322). If the loss of blood has been considerable, intravenous injections of physiologic salt solution may be very beneficial.

**Literature.** Eckart, W. f. Tk., 1899, 312.—Franke, B. t. W., 1901, 179.—Labat & Cadéac, *Rev. vét.*, 1884, 105.—Sequens, *Vet.*, 1896, 236.—Utz, B. Mt., 1873, 116.

**Rectal Hemorrhage of Cattle.** (Hemorrhagic inflammation of the rectum, "Rücken-" or "Lendenblut," [German].) Haubner has described a hemorrhage from the rectum in ruminants, particularly in cattle, which is sporadic or enzootic in character; its anatomical cause is claimed to be a moderate inflammation of the rectum brought about in well-fed animals by the ingestion of irritating acrid vegetable feed.

Holterbach has recently reported several cases of this affection in young cattle of poor owners, also in larger herds. He observed at the same time numerous cases of rectal hemorrhage in dogs.

The **symptoms** are diminished appetite, depression, stiffness of the back, and tenesmus. After one to two days, dry firm feces, mixed with blood, are voided with an effort; sometimes pure blood. The rectum is hot and swollen. In spite of an elevation of temperature (up to 40.2° C.) there is no change of pulse or respiration. The disease usually ends in recovery within two to five days; it may, however, exceptionally lead to a fatal termination.

The **treatment** recommended by Haubner, consists in the administration of salts, and in careful irrigation and emptying of the rectum with mucilaginous fluids, or dilute vinegar and cold applications to the region of the sacrum. Holterbach gives, every two hours, a tablespoonful of the following mixture: Ferri sulphat. sicc. pulv. 150. gm., Sal. Carolin fact. pulv. 300. gm., Pepsin Witte. Germ. 30. gm.

**Literature.** Haubner, *Landw. Tk.*, 1902, 91.—Holterbach, B. t. W., 1908, 81.

### 13. Acute Intestinal Catarrh. *Catarrhus intestinalis acutus.* (*Katarrhalische Darmentzündung* [German]; *Enteritis catarrhalis acuta.*)

**Etiology.** The etiology of acute intestinal catarrh is on the whole identical with that of acute gastric catarrh (see page 285). Errors of diet are concerned preferably in the production of primary intestinal catarrh; these usually produce their bad effect upon the stomach, but they subsequently also excite an inflammatory process in the intestines. Feed which is improper from any cause or taken in large amounts and food which is easily fermenting or irritating are equally noxious. The affection of the intestines is caused either by an extension of the inflammatory process from the stomach to the intestinal mucosa or because the substances causing inflammation are passed unchanged to the intestines, where they exhibit their inflammatory properties. Dahne saw several cases of acute intestinal catarrh

with grave general symptoms after the ingestion of Swedish clover which had grown abundantly and luxuriantly on a pasture. Cattle often show signs of acute intestinal catarrh with profuse diarrhea after the ingestion of green leaves of sugar beets.

Microorganisms frequently play an important part in the production of the affection. Intestinal catarrh is produced in this manner after the ingestion of rotten or sour feed, as for instance sour beer draffs, after the ingestion of meat from sick animals, which is infected with pathogenic bacteria. Uebele claims that dogs sometimes become sick with acute intestinal catarrh if they receive exclusively horse meat which is otherwise unobjectionable. These attacks may even run a fatal course, and it is claimed that the toxic element can be demonstrated in the bouillon from such meat and that it may be made innocuous by sheep or cattle fat.

Whether bacteria which are usually present in large numbers in the intestines, particularly in their posterior portion, may be the cause of disease of the intestines cannot yet be stated definitely. Observations made in human medicine make it very probable that bacteria that are usually harmless may produce inflammatory processes in the intestines of animals if they have become more virulent or if the epithelial covering of the mucosa has become weakened.

Very fibrous vegetable parts which are not, or only with difficulty, digested, as well as ingested sand, sometimes injure the mucosa, while the great majority of laxatives will only produce a catarrhal condition in the intestinal mucosa.

External and internal cooling of the body does not only cause energetic peristalsis by reflex irritation, but it may also produce an intestinal catarrh; this is evidently the cause of the frequency of this affection during the colder season (pasturing on frosted meadows, feeding on frozen potatoes, beets, the ingestion of ice-cold water), while the not uncommon occurrence of the disease during the warmer season (summer diarrhea) is probably usually due to the ingestion of impure water. Working oxen sometimes develop short attacks of intestinal catarrh during the summer if they drink too much water while heated (Cruzel).

As further causes must be mentioned, intestinal worms, intestinal calculi, desiccated feces, the irritating effect of which may be further increased by the presence of bones and other foreign bodies. Catarrh of the rectum may be due to improper manipulation in examination and treatment.

An infection must be assumed to be the cause of the affection if in the absence of errors of diet, numerous animals of a large herd sicken within a short period of time, as occurs frequently among herds of swine (Aronsohn, Esser, authors' observation). In young fowls we observe an enzootic intestinal catarrh which is probably due to infection (Klee).

Intestinal catarrh is often observed to be **secondary** to acute infectious diseases; it also presents itself almost con-



stantly as one of the symptoms of septicemia. Acute intestinal catarrh is particularly frequent in the different septicemic diseases of fowl. (See Vol. I.)

**Anatomical Changes.** Sometimes throughout its whole extent, sometimes in some parts only, the intestinal mucosa is reddened, swollen and abundantly covered with mucus; it sometimes also shows small hemorrhages. The submucosa is in a condition of edematous infiltration. The villi are likewise swollen; they are crowded together and give a velvety appearance to the intestinal surface. The solitary and agminated follicles are swollen and sometimes discharge a purulent mass on pressure, or their surface has become changed into a round ulcer. Especially in the large intestine the mucosa shows a fine bran-like deposit indicating necrosis of the epithelial cells. The feces are fluid or thin; even in the small intestines they are mashy, very fetid and contain threads of mucus, pus, blood. The mesenteric glands are in a condition of acute swelling.

**Symptoms.** The clinical picture of acute intestinal catarrh varies according to whether there is also catarrh of the stomach or whether the intestinal affection has been primary. If there is also catarrh of the stomach the symptoms of the latter predominate, especially lack of appetite and listlessness, and particularly at the beginning.

The most prominent symptoms of acute intestinal catarrh consist in changes in defecation and in the character of the feces. In herbivora, constipation or retarded defecation is often observed for days and occurs in consequence of the preceding catarrh of the stomach. The feces are desiccated, burntlike. In horses there are smaller, dry, hard lumps covered with a shining crust; the feces of cattle appear tortuous or lumpy, coated with a brown layer or with mucus; the small manure of sheep is lumped together by mucus; the feces of swine show lumps adhering to each other.

Carnivora suffer from constipation only for a short time after a few hours diarrhea comes on; the latter is rarely absent in carnivora, more often in herbivora, because in them the affection may be confined to the small intestine and in the large bowel the feces may become desiccated to their normal consistency. However, if herbivora suffer from more severe catarrh or if the large intestine is likewise involved, diarrhea is present and it is one of the most important symptoms of inflammatory processes in the intestines. The feces are now thin-mashy or entirely fluid; they contain poorly digested food stuffs, also frequently mucus and shreds of the epithelial lining, sometimes streaks of blood. They are very fetid, and usually have an acid reaction. Feces of this nature contaminate the region around the anus; they are frequently dropped in large amounts with signs of tenesmus; later involuntary passages may take place. When

the animals defecate the mucosa of the rectum sometimes prolapses and later there is a relaxation of the sphincter muscles with gaping of the anal opening. Fowls' droppings are at first softer, light gray, later perfectly fluid, yellowish or greenish, fetid and they soil or mat the feathers in the neighborhood of the cloaca.

The abdominal circumference either remains normal or is diminished; in other cases it becomes larger. Palpation sometimes elicits pain, especially upon pressure in the region of the small intestines. The intestinal sounds are more lively; they are often audible continually, very loud, rumbling, so that they may be heard for a distance of several feet; they are markedly metallic. Fetid flatus sometimes escapes from the anus with loud sounds.

Convulsive contractions in the intestines of the horse, occurring at short intervals, cause colicky pains which increase after the ingestion of food or drink, and they manifest themselves differently in various animals. Horses may show milder or more severe colicky pains. Cattle manifest pain by dull roaring, groaning, kicking with their hind legs towards the abdomen; dogs hide themselves, lie curled up, howl loudly from time to time, jump up suddenly and run backward and forward; hogs hide in the straw, grunt loudly and frequently roll about; fowls do not show any manifestations of abdominal pain.

The urine is diminished, dark in color, and in carnivora furnishes a rich deposit of urates; its reaction in herbivora is acid even when the appetite is preserved; indican is increased.

The acid reaction of the urine in herbivora depends upon an increase in phosphates. If there is lack of appetite, the starving animals decompose their own proteids. Fröhner believes that the acidity of the urine in herbivora with good appetite depends upon the fact that the contents of the small intestines, which have become acid in consequence of lively fermentation, dissolve a larger amount of the earthy phosphates contained in vegetables, these are absorbed in increased amount by the blood and have to be excreted in increased amount by the kidneys.

The temperature is frequently somewhat elevated. In more serious cases and in severe diarrhea the heat is unequally distributed at the periphery; the pulse is accelerated and the patients are listless. Thirst is usually increased.

If the intestinal catarrh is severe and if diarrhea continues for any length of time, prostration sets in rapidly. The animals now lie motionless on the floor or they are hardly able to stand on their feet; the eyes sink in, the glance becomes lusterless, the hair rough; fowls drop their wings, stand or sit long in the same place; the exhaled air is bad and the whole body has a similar fetid smell. The abdomen is drawn in and tender to pressure; the sphincter ani is strongly contracted or, on the contrary, relaxed; the temperature and the pulse become subnormal, the heart beat becomes hammering, and death now soon ensues.

The clinical picture just described varies in some respects, according to the exact seat of the affection.

If the **small intestines** alone are affected (*Ileitis et jejunitis catarrhalis acuta*) diarrhea may be absent and the feces may appear fairly normal; the intestinal sounds are more lively than under normal conditions, especially in the region of the small intestines; violent pains may occur frequently. Disease of the duodenum may be assumed if there is an icteric discoloration of the mucosæ and if bile pigment appears in the urine.

In **catarrh of the large intestines** diarrhea is rarely absent; symptoms of colic, however, may be absent or quite mild; the intestinal sounds are lively and loud flatus is abundantly discharged from the rectum.

**Catarrh of the rectum** is characterized by frequent, scanty defecation with tenesmus; the feces contain numerous shreds of mucus, which coat the lumps and masses like membranes; the feces are also frequently streaked with blood. On being introduced into the rectum, the finger or hand feels convulsive contractions of the sphincter muscle which later on relaxes, and this leads to prolapse of the rectal mucosa which feels hot and slippery; the introduced finger or hand becomes covered with mucus or blood. By the rectal speculum a markedly reddened mucosa becomes visible.

**Course.** Mild cases last from two to three days, and then the slight disturbances disappear. There are frequently short attacks of restlessness; after the expulsion of gases there are several fluid stools, then marked improvement occurs leading to rapid recovery. In a majority of cases, however, the disease is of longer duration; it lasts about a week and the animals are weak for some time, even after the disappearance of the symptoms. If the intestinal catarrh is more severe, the animals become more and more exhausted in consequence of obstinate diarrhea and a fatal issue may occur in the second or third week. If the disease lasts still longer it terminates in chronic catarrh of the intestines. There are also very grave cases which show profound prostration and grave intestinal disturbances from the start and which end fatally within three to four days. Cases of this kind are observed particularly after the ingestion of spoiled feed or putrid meat and they must really be classified as deeply penetrating inflammations.

A frequent complication of intestinal catarrh is gastric catarrh; its symptoms generally precede, but occasionally follow, those of intestinal catarrh.

**Diagnosis.** The diagnosis meets with difficulties only in the absence of diarrhea, but a correct diagnosis is suggested by lively intestinal sounds, in connection with symptoms of colic, possibly with icterus, and by the fact that the general disturbances and those of the pulse are usually not considerable. Inflammation of the intestines usually leads to grave disturbance of the general condition from the start, weak and frequent pulse,



obstinate lack of appetite, and the impossibility of influencing the disease favorably by treatment.

Since intestinal catarrh may be due to acute infectious diseases and to intestinal parasites, these conditions must be considered in the diagnosis.

**Prognosis.** Primarily uncomplicated intestinal catarrh is dangerous only in very young or very old animals; in these it may occasionally lead to death; as a rule the disease ends in recovery and rarely leads to chronic catarrh. Of unfavorable prognostic significance are elevation of temperature, very severe pains, marked prostration, obstinate diarrhea; these symptoms point to secondary inflammation of the intestines.

Infectious gastro-intestinal catarrh of hogs usually disappears, under purely dietetic measures within one to four days, sometimes only towards the end of the first week. Fatalities do not occur as a rule; Aronsohn, however, reported a mortality of 1 to 2%.

**Treatment.** The main points in the treatment of acute intestinal catarrh are the establishment of favorable hygienic conditions and the proper regulation of diet. The sick animals should, above all, be kept quiet in a warm, dry place where they can, if necessary, be protected against loss of heat by being covered with blankets. The selection of such a place is particularly important in young and small animals, including rabbits and fowls. The feeding ought to be done in the most cleanly manner and the feeding vessels should be cleansed scrupulously before each meal. The parts of the body which may become soiled in defecation must be cleansed, the feces must be removed promptly and frequently from the neighborhood of the sick animals, and an admixture of feces with feed must be strictly prevented. The buccal cavity of the animals should be wiped out several times each day with pure or somewhat acidulated water.

The regulation of the diet should protect the inflamed mucous membranes against further irritation and should attempt to furnish as little culture-material as possible to the microorganisms which rapidly increase in the abnormal intestinal contents. Animals should, therefore, receive a starvation diet for one or two days, if they come under treatment at the first stage and if their state of nutrition permits of the withdrawal of food. All irritating, acrid, undigestible food must be avoided and food must in general be given only in small doses for each meal. Herbivora may receive aromatic, but not fresh, hay (perhaps scalded), grains softened in warm water, crushed oats or barley (best mixed with water and salts), flour or bran mash and, in case the diarrhea is severe, mucilaginous soups of oats, barley, linseed cakes; for ruminants in addition boiled beets and potatoes. Raw feed cannot, as a rule, be withheld entirely, since

the animals will then refuse all food. Diarrhea occurring in cattle after feeding green sugar beet leaves can be stopped rapidly by giving rough feed, particularly hay in the morning as an alternating meal. Sick swine must be fed with flour, rice flour, germinated malt, linseed, bread soups, roasted oat or barley flour, roasted chestnuts, rice, sago, boiled oatmeal with fat, meat broth with the yolk of eggs or a variety of artificial foods (see page 291). After the diarrhea has been stopped, finely chopped meat or fowl meat may be added to the enumerated food stuffs or the patients may receive a mash of crushed barley, rice, etc., to which has been added some fat and salt. Rabbits should receive roasted oats, young twigs of oaks or willow, also burnt flour soup, thick flour paste with roasted rye bread. The following are adapted for fowl: millet, rice, corn and other grains, boiled or worked into a mash with the addition of powdered chalk; also roasted and finely crushed barley, and infusion of half an ounce of oatmeal boiled in one quart of water, or one part of linseed boiled in twenty parts of water; parrots do well on chocolate or on bread moistened with claret. The ingestion of water must be limited and the animals must not receive it cold, but water which has been standing for some time.

Since acute intestinal catarrh generally follows errors of diet, the removal of the gastro-intestinal contents is indicated; this diminishes the amount of the irritating material already in the gastro-intestinal tract, removes a culture soil for excessively multiplying intestinal bacteria and to a certain extent brings about a disinfection of the intestines. If the noxious material is probably still in the stomach then lavage of the latter or the use of emetics may be indicated. The treatment of disturbances of gastric functions, if at all present, must follow the principles laid down above (see page 291). To remove the noxious or abnormally fermenting contents of the intestines mild laxatives should be employed; the best drug is probably castor oil 250-500 gm. to horses; 500-1000 gm. to cattle; 50-200 gm. to calves, foals, sheep and goats; 50-100 gm. to hogs; 15-50 gm. to dogs; 5-20 gm. to cats; to rabbits and birds 5-15 gm. To horses and cattle castor oil is administered with the double dose of hot water or with the same or the double dose of a bland oil (oil of sesame, oil of poppies, etc.); to the dose for horses may also be added 50-70 grams of ether; this makes the oil light-fluid; for dogs and cats the oil may be emulsified with one-fifth gum arabic and five to ten parts of water or with aqua menthæ, tinctura aurantii, succus citri, etc., or in gelatin capsules (3-10 at 5 grams each); to the hog in the form of an electuary with licorice or honey; for fowls a mixture with equal parts of water should be injected directly into the esophagus with a syringe armed at its tip with a flexible tube; or small pieces of stale bread soaked in castor oil are introduced into the pharynx of fowls (Schlampp). According to the experiments of F. Müller calomel has a laxative effect only in dogs (0.3-0.4 gm.), cats (0.1-0.15 gm.), rabbits (0.2

In catarrh of the rectum, particularly in carnivora, the internal treatment indicated should be combined with rectal injections with disinfectants and astringent drugs (argentic nitricum 1-5:1000, alum, tannic acid, sulphate of iron in 1 to 2% solutions, also starch in water, perhaps with some tincture of opium, or with the use of suppositories.

**Rectal injections** are given with a thick-walled rubber tube, connected with a funnel or an irrigator. The fluid should be lukewarm and it should be introduced under a low pressure so that the intestine is not made to contract. In this manner large amounts of fluid may be introduced into the rectum. Before giving an injection, the feces should be removed if possible. In fowls a syringe with a dull nozzle may be used.

Moderate and even heat is also beneficial and reduces the peristaltic movements and in this manner acts against abdominal pain. Hence warm fomentations on the abdominal wall are indicated; also warm infusions given internally. Fowls are often influenced very favorably by hot sand baths (60-70° C.) (Klee).

To counteract the severe weakness which often comes on in young animals rubbing, warm packs, subcutaneous injections of caffeine and camphor are beneficial. Most reliable, however, for this purpose is the intravenous, subcutaneous or intrarectal injection of physiologic salt solution. This latter is best brought to body temperature, after having previously been sterilized, and is injected with aseptic precautions under the skin or into a vein. If injected in this manner, one might also add to the solution 2 to 3% of grape sugar. The intravenous or subcutaneous dose for large animals is 4-5 quarts, 2 quarts for medium-sized animals,  $\frac{1}{4}$ - $\frac{1}{2}$ -1 quart for smaller animals.

**Literature.** Aronsohn, B. t. W., 1898, 110.—Braun, Kaninchenkrkh., 1907, 25.—Esser, A. f. Tk., 1901, XXVII, 306.—Gottschalk, D. t. W., 1909, 497.—Haag, W. f. Tk., 1907, 906.—Hentrich, Z. f. Vk., 1905, 59.—Klee, Geflügelkrkh., 1905, 50.—Vet., Jhb., 1906, 350.—Kramell, Z. f. Vk., 1899, 319.—Müller, Klin. Unters, über Wert und Wirkung des Kalomels. Diss. Giessen, 1908.—Schlampp, Therap. Technik, 1907, II, 1. Hälfte.—Uebele, Therap. Handlexikon, 1910.

#### 14. Acute Gastro-Intestinal Catarrh in Young Animals.

**Occurrence.** <sup>4. 5.</sup> The disease occurs most frequently in calves and lambs, more rarely in foals and still less frequently in other domestic animals.

**Etiology.** The gastro-intestinal organs of young, particularly of sucking animals, are very sensitive and become affected even after slight errors in diet. Saliva is secreted only to a small extent; the epithelial covering of the gastro-intestinal tract is tender and very sensitive, the muscularis is unable to make any greater efforts; the basal cells of the gastric glands are poorly developed, the gastric juice contains comparatively little pepsin, while the pancreas does not secrete any amylolytic



enzyme for some time after birth. All these conditions fully explain the sensitiveness of the gastro-intestinal tract of sucklings to noxious influences. In ruminants there is the further cause that the fore-stomachs are not yet functioning and the fluid food enters the abomasum without any preliminary preparation. This fact becomes particularly important in young ruminants which have been weaned too early.

Since sucklings, as a rule, receive only milk, an improper composition of the latter is generally the cause of the affection. The character of milk is rarely influenced unfavorably by a too abundant feeding of the mother animals with green feed or clover hay. Much more dangerous is the milk after insufficient feeding with watery, non-nutritious, spoiled feed. Certain component parts of the feed, poisonous, oily resinous plants, and certain drugs, particularly laxatives, find their way into the milk and exert a disease-producing effect upon the digestive organs of the sucklings. The milk of cows which receive an abundance of distillery slop or oil cakes may produce gastro-intestinal catarrh in calves.

Certain diseases of the mother animals produce changes in the functions of the mammary glands. Most dangerous in this respect are the acute infectious diseases and still more the inflammatory diseases of the udder. In the course of acute infectious diseases the milk, which is also changed in its composition, frequently contains toxins, and in mastitis certain pathogenic bacteria may coagulate the milk in the udder, or this may have undergone slimy or other changes. Hence sucklings almost invariably become sick if the mother animal suffers from acute parenchymatous or any other form of mastitis, from foot-and-mouth disease, smallpox, epizootic aphthæ, or tuberculosis of the udder. Excessive exertion of the mother animal likewise disturbs the proper secretion of milk.

Disease of young animals is frequently seen in artificial feeding with milk, when the attendants lack in proper cleanliness. Milk is a favorable soil for a variety of bacteria and will easily decompose, the organic acids then formed causing catarrhal processes in the gastro-intestinal tract of young animals. It must also be considered that boiling destroys certain ferments contained in the milk which assist the functions of the gastro-intestinal tract; still more dangerous are milk substitutes; these contain flour almost without exception and are easily decomposed; unsatisfactory is likewise skimmed milk to which flour, bread, etc., have been added.

The ingestion of too large amounts of otherwise unobjectionable milk often produces digestive disturbances. A mistake is sometimes made in allowing young animals to suck only at long intervals, for instance morning and evening; under these conditions the stomach is not able to work up properly the excessive amount of ingested milk. On the other hand, a mistake may be made in the other direction, the young animals may be

permitted to suck too often and they may overload their stomach in this manner.

Finally gastro-intestinal disturbances may be brought about because the young animals have not been permitted to suck the colostrum which removes the meconium from the newborn. Frequently a catarrhal affection of the gastro-intestinal mucosa is due to marked cooling of the body in consequence of staying in cold, damp, drafty barns, or in consequence of sucking from a cold udder, or staying in the open air during cold weather or during rain.

Improper weaning may also produce the affection if only dry feed is given at once to the weaned young. The gastric mucosa is excessively irritated by the coarse feed and at this period the fore-stomachs do not yet function properly in ruminants.

In weaned animals the disease may also be due to the same factors which cause it in adults (see pages 285 and 325).

Bacteria probably also play a rôle in the production of the disease; these micro-organisms are normally present in the intestinal tract and they may be enabled to display pathogenic properties in consequence of errors of diet, or bacteria, which are pathogenic from the start, may be ingested with the food stuffs. The bacillus coli communis appears able occasionally to attain pathogenic properties. (See vol. I.)

Sometimes intestinal parasites may produce a catarrhal inflammation of the gastro-intestinal mucosa.

**Anatomical Changes.** Post-mortem examination frequently shows rather insignificant changes such as hyperemia, a moderate amount of swelling of the gastro-intestinal mucosa; in other cases there may be small hemorrhages, swelling of the follicles, sometimes also superficial ulcerations.

**Symptoms.** The disease usually begins with a diminution of appetite and with a certain degree of lassitude after which febrile symptoms come on. The sucklings do not hunt up their mothers; if they are taken to her they suck little and without relish; weaned animals take little food or none at all. Sometimes vomiting occurs and a marked improvement is then noticeable.

After a short time diarrhea comes on and this remains the prominent feature of the clinical picture and it may even be the very first symptom. In the beginning the feces are of normal color, but somewhat soft, later on they become thinner and thinner, finally perfectly thin-fluid and are sometimes voided in a stream at short intervals, under tenesmus. At this time the feces are yellowish or yellowish-gray, distinctly acid in reaction and of a disagreeable, penetrating, sour smell; they contain white or gray flocculi, lumps (fat-droplets, fat-crystals and bacteria), sometimes bloody streaks, or they are even uniformly reddish. The animals lie curled up on the floor or stand up with their back curved; the legs are drawn under the abdomen and the patients exhibit manifestations of abdominal pain which is generally severe.

The thin-fluid feces soil the neighborhood of the anus; as a consequence the hairs fall out and eczema and intertrigo develop.

The abdomen appears either drawn in and sunken in at the flanks or it is, on the contrary, bloated moderately; the latter condition is seen particularly in ruminants. The intestinal sounds are sometimes exceptionally loud and rumbling. Palpation in the region of the stomach elicits tenderness rarely if at all (the abomasum in ruminants).

If diarrhea is obstinate debility and emaciation rapidly appear. The appetite is now entirely lacking, while thirst may be intense; the eyes sink in, the fur becomes scrubby, the skin is dry or covered with a clammy sweat, the whole body emits a disagreeable nauseating odor. Finally the animals are hardly able to stand on their feet; they lie apathetically on the floor, the peripheral portions of the body become as cold as ice, involuntary movements of the bowels occur and death takes place in syncope or with convulsions.

**Course.** In a majority of cases the disease ends in recovery. The diarrhea stops after a certain time, occasionally after a few hours; the appetite improves, the animals become more lively and generally recover rapidly, even in those cases where the affection has lasted for some time. Grave cases lead to death in three to five days, occasionally, however, they drag along for several weeks and a catarrhal pneumonia is then usually the terminal affection. Aphthous stomatitis is a frequent complication in lambs.

**Diagnosis.** The disease may be confounded with dysentery of sucklings; the latter, however, always attacks the animals immediately after birth or at the utmost during the first few days of life; it takes a much more unfavorable course and betrays clearly its infectious character, while simple gastro-intestinal catarrh appears sporadically, and even if there are a number of cases, one is able to demonstrate the common external factor in its production.

**Prognosis.** The younger the sick animals, the longer the disease lasts and the less favorable is the prognosis. The latter also depends upon whether it is possible to remove the noxious causative factors.

**Treatment.** The first attempt at treatment must be the regulation of the diet of the sick animals; if they are sucklings, the diet of the mother animal must likewise be attended to and errors of diet or external unfavorable conditions have to be corrected speedily. In case the young animals have to be brought up by hand, it is advisable to feed them with pasteurized milk, formalin milk (1 to 25,000) or perhydrazine milk. The detrimental effect of feeding milk may be diminished by the addition of



lime water (a teaspoonful to a quart of milk); its often markedly bloating effect may be removed by the addition of boiled oatmeal. Lafitte saw unfavorable results from the exclusive use of fresh whey. Sucklings should be allowed to feed three or four times per day and if necessary should be fed by a healthy foster-mother. The diet of weaned animals must be regulated in the same manner as is recommended in intestinal catarrh of adult animals (see page 330). The barn should be kept uniformly warm and the place where the young animals are must be cleansed very thoroughly. The teats of the mother animals should also be cleaned before the young are allowed to suck.

The medicinal treatment is similar to that used in dysentery of the newborn (see Vol. I) or to that used in gastro-intestinal catarrh of adult animals (see page 331).

In calves favorable results have been obtained by the administration of salicylic acid with tannic acid  $\bar{a}\bar{a}$  1-2 gm. three times a day in camomile tea). Schwarzmeier recommends for foals tincture of opium (up to 5. gm. pro dosi) with tannic acid in whiskey; Schley, for calves, a  $\frac{1}{2}\%$  solution of nitrate of silver. Sometimes washing out of the rumen (see page 274) may become necessary (Imminger). Eber has brought about a cure in two calves, after collapse had occurred, by the subcutaneous injection of two quarts of physiologic salt solution (Na Cl 0.8% carbonate of sodium 0.25%). The absorption of this solution goes on very rapidly, especially if aided by massage. Kronacher had similar favorable results with the rectal application of physiologic salt solution.

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## 15. Catarrhal Intestinal Colic. Enteralgia Catarrhalis.

(*Einfache Reiz-, Krampf-, rheumatische or Erkältungskolik*  
[German].)

What is generally called rheumatic or convulsive colic is a mild, short attack of acute intestinal catarrh with colicky pains.

If a special chapter is here devoted to this affection, this is done because the affection is frequently treated separately in veterinary literature as a form of colic. The French writers include the disease in the great group of "*congestions intestinales*," to which also belong the disturbances due to thrombosis of the mesenteric arteries. The term "convulsive colic," "*Krampfkolik*" (German), appears objectionable because all genuine colicky pains are due to convulsive contractions of the intestines.

**Occurrence.** Symptoms of colic in the horse undoubtedly are due most commonly to a transitory acute intestinal catarrh. Thirty-five per cent of the colic affections seen in the Budapest clinic are due to this affection and it is the most frequent type of colic among breeding and cavalry horses. Cattle and hogs are likewise sometimes subject to this disease.

**Etiology.** The causative factors are the same as in acute intestinal catarrh (see page 325). In a certain proportion of cases external or internal cooling of the body plays a rôle; (drenching, standing in cold weather in the open air when heated, the ingestion of excessively cold water or of cold frosted feed). Catarrh from these causes is, however, rarer than catarrh due to errors of diet. The loud intestinal sounds which are considered characteristic for the disease, the so-called "cramp sounds," clearly point to lively fermentative processes in the intestinal tract with an increased formation of gas; the disease occurs just as frequently during the warm season, when the animals are less exposed, as during cold weather. Cavalry horses, which, as a rule, receive little rough feed and which in consequence often feed upon the bedding straw, frequently suffer from the disease after the ingestion of mouldy straw (Pr. Mil. Vb.). In cattle, particularly in working oxen, catarrhal intestinal cramps are often caused by the ingestion of large amounts of water (see page 326).

**Pathogenesis.** The irritating substances (gases, fatty acids) or too much cold water cause convulsive contractions in various portions of an irritable intestinal wall, which vary in duration. These convulsive contractions then cause the colicky pains (Bauchzwicken, Bauchgrimmen [German]). After the abnormal intestinal contents have been voided, the convulsive contractions cease, but the peristalsis for some time remains more lively because the intestinal mucosa is in a catarrhal condition.

**Symptoms.** Horses are subject to symptoms of colic which are usually violent; the individual attack lasts from five to fifteen minutes and the animals appear well during the intervals. The intestinal sounds are usually intensified and more frequent, sometimes continuous and occasionally audible, not merely over the cæcum but likewise over the remainder of the intestines. Defecation may be perfectly normal, that is, if only the small intestines are affected, or it occurs more frequently and the feces are soft and contain undigested particles of feed; sometimes there are short attacks of diarrhea. In cattle severe attacks of colic come on suddenly, the abdomen is occasionally somewhat enlarged and the left flank depression is somewhat filled out, although bloating is absent. After half an hour diarrhea sets in and with it a gradual diminution of the pains; the animals usually appear perfectly recovered after a few hours.

**Hogs** become very restless in consequence of catarrhal intestinal pains; they lie down frequently, roll, groan, sigh, or cry out aloud. After the expulsion of flatus and after several defecations they become quiet.

**Course.** In the course of mild cases the attacks of pain last only a few quarters of an hour; the affection rarely extends over six hours, but even after the symptoms of pain and restlessness have disappeared, the intestinal sounds persist for some time. The very mild character of the catarrhal affection of the intestinal wall fully explains the rapid recovery after the irritating substances have been carried off or the causative factors have been favorably influenced. The course is favorable almost without exception; only very rarely do excessively strong intestinal contractions lead to volvulus.

**Diagnosis.** The appearance of abdominal pain in paroxysms, the absence of an increase of the abdominal circumference in the horse, usually also in cattle and swine, in connection with a lively peristalsis with commonly more frequent defecation, the absence of general disturbances, the negative result of an exploration per rectum are characteristic for the affection under discussion. Embolism of smaller intestinal arteries occasionally also causes abdominal pain. A differential diagnosis is only possible, and then not always, if the history shows periodic attacks of abdominal pain without any external cause, with a normal character of the feces, and when rectal exploration demonstrates thrombosis of the mesenteric artery, in which case one may, of course, assume an embolic closure of some small intestinal vessels.—Abdominal pains due to uterine contractions, which in mares come on towards the end of gestation and are due to energetic fetal movements, do not lead to an intensification of the intestinal sounds and the fetal movements can easily be perceived.—Strangulation or invagination in cattle can be excluded from the history and the findings.

**Treatment.** Warm applications upon the abdomen and rectal injections with warm water have a tendency to stop the cramps of the intestines. Warm applications may be made in such a manner that two sacks are sewed together, dipped in water of 40° C., placed upon the abdomen and covered with a dry blanket. The application must be renewed every ten minutes. If in horses the abdominal pain is very intense morphine (0.3-0.5 gm.) may be given subcutaneously or chloral hydrate per rectum. To expel the decomposing intestinal contents rapidly mild laxatives may be given which are not liable to lead to cramps, neutral salts, castor oil, combined with ether for horses; the diet should be regulated as in acute intestinal catarrh (see page 331).



**Abdominal Pain in Uterine Contractions.** Highly bred mares frequently show symptoms of colic in the advanced stages of gestation, especially about the 8th month of pregnancy and during the last weeks before parturition, these depend upon a mechanical irritation of the uterus by the moving embryo and upon uterine contractions caused by it. Predisposing causes are changes in position of the embryo, external dull force to the abdomen, overheating followed by the ingestion of cold water, the ingestion of spoiled feed.

The symptoms are similar to those of catarrhal intestinal cramps, but the hand placed upon the abdomen may feel the kicks of the embryo and they may become visible on the abdominal wall. The affection lasts from 1 to 3 hours and then disappears suddenly; in some mares the painful attacks recur within short intervals, sometimes every second day, even every 6 to 8 hours, especially shortly before parturition; (Schleiffer Vet. 1894, 229).

For treatment subcutaneous injections of morphine, also the internal administration of camomile tea are indicated.

## 16. Chronic Intestinal Catarrh. *Catarrhus intestinalis chronicus*.

(*Enteritis catarrhalis chronica*.)

**Etiology.** Primary chronic intestinal catarrh, which is comparatively rare, usually arises from an acute intestinal catarrh if the latter has lasted for some time or if the inflammatory influences have acted for a considerable period upon the intestinal mucosa. The causative factors are the same as those of acute intestinal catarrh (see page 325), the most common cause, however, is improper feeding and the continued ingestion of improper or spoiled food.

According to the statements of Dieckerhoff, there occurs in foals, also in cattle, sheep, swine and goats, during the first two years of life, a chronic disturbance of the function of the intestinal mucosa, which leads to progressive emaciation and frequently to death. Dieckerhoff has named this disease "*Darrsucht*" (German) or *Tabes intestinalis* (intestinal wasting away) and has separated it from the disease *Tabes mesaraica*. (See Vol. I.) The etiology of the disease is unknown, its causes are probably various emaciating internal diseases. According to Glage, this *Tabes intestinalis* in foals is in reality an infectious sclerostomiasis (q. v.).

Dawson has described a disease observed in the United States under the name of Infectious Asthenia in chickens two to six months old, which is caused by a bacterium of the colon group (*bacterium astheniæ*) and characterized by a profound duodenal catarrh. The same disease has occasionally been observed in Germany by Kitt.

Some of the cases described by Dieckerhoff and Harms as chronic gastric catarrh of cattle, and a disease described in France as "*diarrhée chronique*," "*boyau tendre*," "*dysentérie*," should more properly be classified as "*enteritis paratuberculosis* (Bang). (See Vol. I.)

More commonly chronic intestinal catarrh is a secondary affection. Such secondary chronic intestinal catarrhs are seen

in chronic passive congestion of the intestines due to chronic diseases of the heart and liver, very rarely in certain cases of thrombosis of the mesenteric artery, in chronic infectious diseases (tuberculosis, pyobacillosis [Grips, Glage & Nieberle]), chronic hog erysipelas (Eisenmann) and in chronic constitutional diseases. Intestinal worms likewise frequently produce the picture of this disease.

**Anatomical Changes.** The intestinal mucosa usually appears dark purplish to brown red, sometimes slate colored, in consequence of the presence of numerous blackish points, and thickened, while the glands of the propria are enlarged, tough, or changed into cysts. Sometimes, however, the mucosa is thin, pale, and shows depressions in the place of the solitary and agminated follicles (so-called areolation). Occasionally there are formed round, thick but shallow, later on deeper or confluent irregular ulcerations with undermined and tough margins. After healing, these ulcers leave radiating cicatrices. After perforation of such ulcers purulent ichorous peritonitis occurs in their neighborhood, and the affected loop of intestine becomes adherent to neighboring loops, to other abdominal organs or to the abdominal wall.

In asthenia of chickens the intestinal contents appear to be only mucus, the duodenal mucosa is reddened at its prominent points and the cadaver is much emaciated.

**Symptoms.** When chronic intestinal catarrh has existed for some time, the nutrition of the animals suffers to a high degree. They are listless and their ability to work is much diminished; later on their condition becomes very poor, the skin is dry and non-elastic, the fur rough and lusterless, the mucosæ are pale. The appetite is variable, sometimes the animals eat an enormous amount, sometimes they refuse food for days. The abdomen is drawn in although a moderate chronic bloating occurs occasionally, particularly in cattle. Defecation occurs at irregular intervals, constipation lasting for several days alternates with violent diarrhea; sometimes the feces are dry and they are dropped after considerable intervals, at other times they are thin mushy or fluid, containing poorly digested particles of food, shreds of mucus, lumps of pus, and they are sometimes streaked with blood; they are often very fetid. Occasionally masses of mucus without any feces are voided.

From time to time, particularly when bloating is present, large amounts of intestinal gases are expelled from the rectum. Peristalsis is absent during constipation, very lively during diarrhea. Colicky pains are rare, when they occur, they usually precede diarrhea or appear while it lasts. During lack of appetite in herbivora the urine often becomes acid; but Friedberger & Fröhner as well as Albrecht usually found an alkaline reaction

of the urine under these conditions. Bauer found an increase in indican.

According to Dieckerhoff, the symptoms of intestinal tabes of young animals, are diminution of appetite, partial constipation, occasionally diarrhea (Krönig), emaciation progressing to the point of cachexia, and leading in three to six months to death unless something can be done by a proper change of diet.

In infectious asthenia of chickens one sees listlessness, depression, progressive emaciation in spite of greedy feeding, pale discoloration of the comb and of the wattles, of the throat and ears, and slight constipation. In the further course of the disease the appetite becomes variable. Finally, the cachectic animals succumb after the disease has lasted about three months.

**Course.** Chronic intestinal catarrh may last for months or even years and it may lead to complete exhaustion of the sick animals, after an edema has developed on the extremities and in the lower abdominal region. Death takes place from marasmus. The disease usually takes this course in old, poorly nourished, hard worked animals.

**Diagnosis.** Progressive emaciation, occasional attacks of diarrhea, which rarely last continuously for weeks and months, the presence of undigested particles of feed in the feces are signs upon which the diagnosis rests. As to the localization of the affections the same rules hold good as are laid down for acute intestinal catarrh (see page 329). Lumps of pus in the feces point with great probability to ulcerations.

Considering the fact that chronic catarrh of the intestines is usually a secondary affection, one should always carefully examine all organs and look into the method of feeding in order to find out the primary disease. As such, one must think of chronic infectious diseases, metabolic diseases, helminthiasis, chronic disease of the liver, chronic uremia, and in horses disturbances of mastication in consequence of anomalies of teeth.

The disease called "Darrsucht," according to Dieckerhoff, may be recognized from the fact that the improperly fed young animals of one herd or flock all become more or less sick and emaciate progressively. Tuberculosis has sometimes to be excluded by a properly conducted tuberculin test.

A diagnosis of infectious asthenia of chickens is suggested by the fact that the sick animals are all two to six months old, that cachexia develops progressively in spite of a good appetite, that the course is quite chronic and that there are no pronounced anatomical changes. The differential diagnosis must always consider the possibility of helminthiasis.

**Prognosis.** Chronic intestinal catarrh, whether primary or secondary, must always be looked upon as a serious affection which will often resist the most careful treatment for a long



time or even permanently. The prognosis is particularly unfavorable in old animals and when a proper regulation of the diet is impossible. The prognosis of secondary chronic intestinal catarrh depends upon the primary underlying disease. There is no hope of recovery in "Darrsucht" after cachexia is once present; young animals three to four months old generally succumb to this disease. Chickens affected by infectious asthenia generally die.

**Treatment.** A rational diet (see pages 295 and 330) offers the best means of preventing the development or to stop the progress of chronic intestinal catarrh. The sick animals should be taken out daily and should be used for work only moderately. Animals attacked by "Darrsucht" may recover in one to two month if they are pastured, provided that the disease has not progressed too far.

To regulate defecation, castor oil and the neutral salts are indicated, among the latter particularly Carlsbad salt (for dosage, etc., see page 331). The desired effect can, however, only be attained if the salts are administered to the animals one-half to one hour before feeding, and in moderate doses (130-500 gm. pro dosi, or of the 5% solution, tablespoonful doses). This course must be continued for a long time and kept up for some time after recovery. With the salts may be combined bitters (gentiana, calamus, rheum, oak bark). Constipation must be counteracted by mild laxatives, diarrhea by astringents and mucilaginous medicines (see page 332). Good results have been brought about in horses by the systematic administration of creolin.

A good result may as a rule be expected only in primary catarrh and while the general condition is still fairly good. The authors again point out the possibility that intestinal worms may be the cause of the trouble; cases which have been treated for a long time without any success at all, have yielded in a short time, after an antihelminthic treatment was instituted.

Against infectious asthenia of chickens, Klee recommends the administration of castor oil (in teaspoon doses) or of calomel (0.01-0.05 gm.) until a marked laxative effect becomes manifest. The birds ought to receive further, daily twice per head, 1-2 gm. of the following mixture: Fennel, anise seed, coriander, bark of quinia, each 4 gm.; powdered root of gentian and ginger, each 7.5 gm.; sulphate of iron 2.0. As drinking water the fowls should have a watery solution of sulphate of iron and sulphuric acid (1000:2:2). The healthy animals must be separated and the coops must be disinfected.

**Literature.** Bächstädt, Z. f. Vk., 1908, 159.—Dawson, Anim. Ind., 1898, 329.—Dieckerhoff, Spez. Path., 1892, 11, 313, 1904, 1, 638.—Eisenmann, Monh., 1906, XVII, 97.—Glage, Z. f. Infkr., 1905, 1, 341.—Grips, Glage & Nieberle, Die Schweine-seuche, 1904.—Klee, Vet. Jhb., 1901, 245; Geflügelkrkh, 1905, 26.—Kramell, Z. f. Vk., 1899, 319.—Kröning, Z. f. Vk., 1906, 202.

### 17. Membranous Enteritis. Enteritis membranacea.

(Socalled "*Darmkrupp*" [German] or *Croupous enteritis*; *entérite cœueneuse, ou pseudomembraneuse ou muco-membraneuse* [CADÉAC] [French].)

Membranous enteritis is characterized by a peculiar, generally superficial, inflammatory process of the intestinal mucosa, with the formation of pseudomembranes, which are composed almost exclusively of mucus.

**Occurrence.** This rather rare affection is observed most frequently in cattle, according to Keilgaard quite frequently in horses, rarely in sheep (Clavel, Lafosse). Among cattle, young, well nourished and pregnant animals are affected preferably if after winter feeding they are pastured during cool weather on luxuriant meadows.

**Etiology.** The nature of the affection suggests infectious material as its cause, while external influences, such as the ingestion of too cold water or the feed supply play a rôle as predisposing factors. Nothing definite can be said as to the nature of the supposed infectious agent. The disease has been observed frequently after the ingestion of rotten potatoes or beets or of absolutely fresh feed.

Whether an occlusion of the intestines or the lack of peristalsis, due to intestinal bacteria, may cause an accumulation of mucus cannot at present be decided. It is, however, a fact that abundant thick masses of mucus collect in the rectum of horses, similar to croupous membranes, if an internal occlusion, situated between rectum and stomach, prevents peristalsis of the rectum.

**Anatomical Changes.** As a rule the jejunum, cecum and colon are uniformly changed; however, the small intestine or on the other hand the large intestine alone may be the seat of pathological changes. The intestinal contents are thin fluid and dirty discolored, mixed with blood in grave cases and very fetid, floating in it are found yellowish to brown, tough, elastic membrane-like or tubular, cylindrical masses. Similar masses are found adhering to the mucosa. The tubular or cylindrical deposits are usually from 1.5 to 3.5 feet long, but they may exceptionally be as long as 35 feet (Arnal). They are composed of several layers, the membranous substance alternating with layers of darkly stained fecal matter. The intestinal mucosa shows reddening in grave cases, also hemorrhages, it is somewhat swollen. In the horse one usually finds changes only in short portions of the large intestine and the pseudomembranes are characterized by their small dimensions.

The microscopic examination of the pseudomembranous deposits show the presence of numerous granules in an exceedingly fine reticulum (Gurlt). Weigert's fibrin

stain never shows any fibrin in the cases examined, and the proper stains demonstrated the presence of mucin (Johne). Chemical tests made by Lassaigue showed that the pseudomembranes consist almost exclusively of condensed mucus; the same results were obtained by Clément who investigated membranous enteritis in horses.

All investigations made have failed to furnish any proof that membranous enteritis is a genuine diphtheritic inflammation of the intestinal mucosa, but they have rather shown that we are dealing with a peculiar purely catarrhal process with an overproduction of mucus, analogous to the membranous catarrh of the stomach of man (Nothnagel). One could not reconcile the usually mild course of the infection with a grave croupous inflammation. The preferably catarrhal character of the process does, of course, not exclude an occasional deeply penetrating inflammation.

For these reasons it appears proper to consider here this form of enteritis and to separate it from true croupous inflammation. This will be considered among the gastro-intestinal inflammations (q. v.).

**Symptoms.** The disease usually begins with disturbances of digestion and mild attacks of colic. The animals do not eat much, they are depressed and ruminate irregularly. One also observes muscular tremor, reddening of the conjunctivæ and an acceleration of pulse and respiration. The secretion of milk is suppressed. The mild attacks of colic last only 12 to 15 hours and then cease for several days. The feces are at first dry; they are dropped less frequently and with increased abdominal pressure. After five or six days, rarely somewhat later, colicky pains recur, the animals walk around restlessly, turn their heads to look towards the abdomen, kick with their feet and drop feces with signs of tenesmus. These are thin fluid and fetid, mixed with gray or yellowish-white membranous, tubular or cylindrical masses of variable, sometimes of considerable length. The solid cylinders or the tubular pieces are either empty or filled with feces and may at first sight be mistaken for pieces of intestines. However, their homogeneous structure and the absence of mesentery and blood vessels prevent such a mistake. After one or two days the symptoms suddenly disappear; however, the feces remain fetid for several days and are mixed with mucus; then the animals recover completely.

Deviations from this clinical picture occur in two directions. In very mild cases animals previously apparently quite healthy, very unexpectedly drop such membranes as described, perhaps with symptoms of a little restlessness, then thin fluid feces, mixed with mucus, then they soon get well again (Lafore, Reynal, Combe). There are, however, on the other hand, cases which run from the start a course with the symptoms of a grave enteritis or these symptoms may develop later on and profuse hemorrhages may occur in the further course of the disease. In cases of this kind there is great prostration, complete lack of appetite, high fever, the pulse cannot be felt, the peripheral parts of the body feel cold as ice and the fluid and fetid feces are more or less hemorrhagic.



In horses, the disease likewise usually begins with restlessness; this soon disappears and the animals then stand apathetically and stagger on standing and walking. In other cases colicky symptoms are at first absent, the disease begins with a diminution of the appetite which soon disappears completely. There is often fever ( $39.5^{\circ}$ - $41^{\circ}$  C.) and always an acceleration of the pulse. In the further course of the disease, colic appears and the feces are profusely covered with mucus; grayish-white, or white opaque, structureless or curled-up long strands or membranes of mucus may also be present free in the intestines or they may be voided with the feces. The disease always ends in the recovery of the animals after a few or several days.

**Course.** The whole course usually lasts from 8 to 12 days, it may be somewhat shorter or on the contrary longer; in the latter case, exacerbations occur during the voiding of the pseudomembranes and remissions at other times. The disease usually ends in recovery; a fatal issue occurs in only such cases as are complicated with grave intestinal inflammation or intestinal hemorrhages; in these the animals may succumb after four or five days. Very exceptionally there may be a fatal obstruction of the intestines by the accumulated pseudomembranes.

**Diagnosis.** The true character of the disease can be recognized only after the appearance of the pseudomembranes; before this occurs it cannot be distinguished from other inflammatory processes of the intestinal tract. The pseudomembranes have a typical appearance and can easily be distinguished from pieces of tendons that are accidentally taken up with the feed, from tapeworms, loops of intestines and other objects.

**Treatment.** The expulsion of the pseudomembranes and of fetid feces may be hastened by the administration of alkalies, especially the laxative salts (see page 331); later on mucilaginous mixtures and astringents are indicated, similar to those recommended in other forms of intestinal inflammation (see page 331). The regulation of the diet must follow the same principles as are laid down in the treatment of acute intestinal catarrh (see page 330).

**Literature.** Clavel, J. du Midi, 1860, 361.—Combe, Pr. vét., 1900, 157.—Delafond, Rec., 1842, 217.—Graziadei, Clin. vet., 1901, 592.—Gurlt, Mag., 1847, 80.—Imminger, W. f. T., 1904, 55.—Keilgaard, Maanedsskr., 1907, XIX, 81.—Nothnagel, Die Krankh. d. Darnes, 1898, 139.—Reynal, Diet., 1860, 87.

**Enteritis Pseudomembranacea of Cats** (croupous enteritis of cats). This is a disease evidently peculiar to cats. According to Zschokke, who first described it, it is most common among young cats and appears epizootically during winter and spring. Cases of enteritis pseudomembranacea have been observed also by Kitt, and more recently by Schmul, who studied the histologic changes of the disease.

Zschokke believes that the probable cause of the disease is a virulent variety of the colon bacillus. This variety, when fed to a young cat,

produced a transitory intestinal catarrh and twitching of the muscles; in another cat it led to a fatal intestinal catarrh. Schmul believes that several kinds of bacteria are concerned in the causation of the disease.

The most important anatomical changes consist in an increase in thickness, diminished elasticity and a greater transparency of the wall of the small intestines; in about one-half of the cases there are deposits on the mucosa; the latter, however, is almost always reddened and swollen. The contents of the small intestines are of a milky turbidity, while the contents of the large intestines are frequently mixed with blood.

The histologic examination shows that the intestinal villi are markedly contracted, the tissues of the intestinal wall are edematous, there is frequently necrosis of the villi, and this may extend even into the serosa. There is also a moderate leucocytic infiltration and likewise moderate hemorrhagic extravasation.

The pseudomembranes are, according to Zschokke, composed of a mixture of epithelia, leucocytes and fibrin threads, but according to Schmul, they are mostly made up of bacteria and do not give either a distinct mucin or a fibrin reaction.

The mesenteric lymph glands usually show acute swelling; the liver and the kidneys are congested.

The symptoms of the disease consist in vomiting and diarrhea and these usually lead to death in one to three days, so that a suspicion of poisoning is frequently created.

Treatment is unpromising, according to past experiences.

**Literature.** Kitt, *Pathol. Anat.*, 1906, II, 61.—Schmul, *A. f. Tk.*, 1907, **XXIII**, 445.—Zschokke, *Schw. A.*, 1900, **XLII**, 20.

## 18. Inflammation of the Stomach and Intestines. Gastro-enteritis.

*Septic, typhoid, mycotic enteritis, intestinal mycosis, intestinal typhoid, dysentery of adult animals, Magenruhrseuche* [German], *Fungus and meat-poisoning, croupous or diphtheritic enteritis, Mycosis seu sepsis intestinalis, gastro-entérite, gastro-entérite dysentérique* [French], *pseudo-typhoid*).

Under the name of gastro-enteritis are comprised all of those inflammatory processes which affect either the stomach or the intestines, but usually both of them simultaneously, and which are quite intense in character, leading to hemorrhage, supuration or to the formation of true croupous membranes due to necrosis. These conditions do not constitute one specific entity, but they have to be treated collectively under a common head, since the necessary investigations are lacking to separate them properly into several specific types.

Severe inflammatory processes in the stomach and intestines may develop upon the basis of a variety of causes, hence the clinical picture varies a good deal. In certain cases the symptoms are quite characteristic for particular uniform types and therefore justify a separate consideration under a special head (see foreign bodies in the stomach, membranous enteritis, coccidial dysentery). A separate consideration is also indicated for inflammations produced in the course of poisoning

(by acids, alkalies, phosphorus, arsenic, lead, mercury, croton oil, carbolic acid, cantharides, lupinae and other plants containing acrid substances. Consult text-books on Toxicology.). Those forms of gastro-enteritis will not be considered here, which occur in the course of acute infectious diseases (anthrax, all forms of hemorrhagic septicemia, hog cholera, rinderpest, influenza, distemper, dog typhoid, etc.).

**Etiology.** In some cases gastro-enteritis develops after a simple catarrh of the stomach or intestines or it is brought about by the same factors which cause the latter. Lüdecke saw a hemorrhagic enteritis in cattle after the feeding of green sugar beet leaves which contained sodium saltpeter. Similar cases have been observed in horses which had died in collapse before diarrhea had set in. With the exception of these types the most frequent causes of gastro-enteritis are spoiled feed (so-called mould poisoning, gastro-enteritis mycotica, mycosis intestinalis).

Herbivora sicken frequently after the ingestion of large amounts of spoiled and mouldy feed. As such may be mentioned rotten and fermenting beets or cuttings of beets, potatoes, to a lesser extent mouldy, moist grain or rough feed, particularly if much contaminated with rust-fungi (see page 183). Marek has produced hemorrhagic enteritis in rabbits with the uredospores of *Puccinia graminis*; Push in the same animals with another fungus (*Tilletia caries*); other authors, however, were not able to demonstrate experimentally the poisonous nature of these moulds. The ingestion of bedding straw and manure is sometimes also dangerous. Water from wells in the neighborhood of cesspools, or water contaminated with animal offal has repeatedly caused numerous affections.

Carnivora very frequently develop a gastro-enteritis (meat poisoning) after the ingestion of infected or spoiled meat. Meat derived from animals which have suffered from certain infectious diseases may exert its disease-producing influence by bacteria contained therein, by their toxins, or by ptomains subsequently formed, or by the combination of all three factors. Most important in this respect are the pyogenic bacteria or rather the diseases produced by such as septicemia and pyemia (puerperal fever, the various forms of wound fevers, purulent arthritis, purulent inflammations of serous membranes, streptococcimastitis, enteritis, etc.). In carnivora the bacteria of meat poisoning (see there) are of importance. Sometimes meat becomes dangerous on account of specific septicemic diseases such as anthrax. In all septicemic diseases the circulating bacteria and their toxins are found with the blood in the muscles and in the internal organs. The dangerous nature of the meat may increase after slaughter because the pathogenic bacteria may increase further.

In 1895 numerous fatal cases of gastro-enteritis were observed, in the southern parts of Hungary, in dogs which had fed upon the meat of swine sick with hog



cholera. It could not be decided in these cases whether the affection of the dogs was due to the microorganisms of hog cholera, to its toxins or to other bacteria. Berger saw hemorrhagic gastro-enteritis in the dogs of a village after these animals had fed upon parts of chickens that had been sick with fowl cholera.

Meat or meat preparations coming from healthy animals may subsequently become poisonous in consequence of contamination with bacteria which may form organic poisons or which may themselves be of a pathogenic nature. Postmortem putrefaction of albumen does not, however, appear to have a particularly deleterious effect (M. Müller).

It is quite possible that bacteria which are important in this respect for meat may come from the feces or from the insufficiently cleaned intestinal wall. This would explain the often very dangerous nature of sausages, because they offer a chance for the chopped-up meat, etc., to become infected from an insufficiently cleaned intestinal wall. Some of the bacteria in question are common inhabitants of the intestines, such as some species related to the *bacillus coli communis* which have been demonstrated repeatedly in cases of meat poisoning.

Putrefaction of albumen leads to the formation of poisonous substances which may undoubtedly occasionally lead to meat poisoning characterized by paralysis of nerves. The most important of these are:

Neurin, a strong poison formed in putrefying meat after 5 or 6 days, probably in consequence of bacterial activity. Symptoms of neurin-intoxication are difficulty in respiration, weak heart, salivation, increased peristalsis, diarrhea, convulsions, finally collapse.

Muscarin is identical with the poison contained in the poisonous fly toadstool; it leads to similar symptoms.

Methylguanidin, another not well determined ptomain, is formed in putrefying horse flesh; it causes convulsions and paralysis of the heart.

Less poisonous are cadaverin and putrescin which cause local inflammations and death only in larger doses, cholin (poisonous only in very large doses) and finally a ptomain isolated by Garcia from putrefying horse flesh.

Pathogenic bacteria differing from those already mentioned and generally not yet well known may get into the intestinal tract with feed that is otherwise perfectly unobjectionable. Such bacteria probably cause dysentery of cattle, which sometimes occurs enzootically among barn fed cattle, rarely on the pasture. The disease attacks almost without exception adult animals. There is also the possibility that inflammations may be caused by the normal intestinal bacteria in animals debilitated by insufficient feed. It is probable, on the other hand, that dysentery in adult cattle and sheep not due to coccidia is frequently in fact a form of hemorrhagic septicemia (see Vol. I).

Under the name of dysentery are comprised in veterinary medicine a variety of deeper inflammatory processes of the gastro-intestinal tract, while in human medicine (aside from tropical amebic dysentery) dysentery designates a specific endemic, aliphtheritic intestinal inflammation caused by the *Bacillus dysenteriae* of Shiga-Kruse. It is possible that a specific, enzootic dysentery may occur in animals, especially in cattle; however it cannot at this time be separated from other forms of gastro-enteritis nor can its relation to human dysentery be determined.\*

\*It has however been shown that there are various types of human bacillary dysenteries caused by a variety of bacilli all belonging more or less to the colon group; such bacilli acting as the cause of dysentery, and not quite identical with the Shiga-Kruse bacillus have been isolated by Flexner and others. (Translators' notice.)

**pneumonia.** If gastro-intestinal catarrh comes on after excessive efforts (long rides, particularly during great heat, long lasting railroad transportation) one may assume that a lowering of the resistance of the organism as a whole had led to a lowering of the resistance of the intestinal wall and had exposed it to the detrimental effect of intestinal bacteria. The intestinal affection coming on within twenty-four hours after excessive work ("Distanzrittkrankheit" [German] of Heuss) has generally a hemorrhagic character and usually ends fatally.

Larger masses of sand which have found their way into the gastro-intestinal canal sometimes cause gastro-enteritis in horses (Mazulewitsch, Wenderniikow and others) and in hogs, (authors' observation) owing to mechanical irritation and to small injuries which open up portals for the invasion by bacteria. In this manner hogs often become affected when they are transported in railway cars the floor of which has been covered with sand.

**Pathogenesis.** Every gastro-enteritis is really due to microorganisms or their toxins; their effect is not confined to the intestinal wall but extends to the entire organism. Bacteria and their metabolic products are readily absorbed in the inflamed intestine and then easily lead to general intoxication or infection. This may occur the more easily since such intestinal inflammations spread to the submucous tissue which is quite rich in lymph channels. (Some bacteria can, even in a healthy intestine, travel into the mesenteric gland and into more distant organs as shown by the investigations of Porcher, Desoubry, and more recently by Bogozinsky, Wrzosek & Ficker). Hence gastro-enteritis is complicated from the start by more serious general disturbances or by affection of more distant organs.

**Anatomical Changes.** The gastro-intestinal mucosa is affected to a variable extent from case to case, sometimes more uniformly, sometimes in patches, especially on the thick rugæ which are of a lively red color, either light or dark with punctate or streaked hemorrhages; there may also be more extensive suffusions of blood. In the cases of gastro-enteritis of dogs which the authors have observed, they found sometimes the mucosa of the stomach, at other times that of the intestines, diffusely discolored dark red and moderately swollen. The gastro-intestinal contents are sometimes hemorrhagic, usually quite fetid. The small hemorrhages in the stomach lead to the formation of small, round, sharply defined erosions (erosiones hæmorrhagicæ). The mucosa and submucosa are edematous, the submucosa of the stomach sometimes shows purulent infiltration (gastritis purulenta sive phlegmonosa). The abdominal serosa is hyperemic (arterial injection).

The solitary and agminated follicles are considerably swollen and the necrotic epithelial cells form a hoar-frosted, ashy or bran-like covering. Necrosis may penetrate more deeply whereupon the various layers of the mucosa become changed into dirty gray, opaque-yellow pseudomembranes; after these are shed sinuous ulcers remain behind, round or large and irregular, bounded by undermined margins, the base covered by necrotic tissue shreds. The bacillus necrophorus leads to the formation of flat ulcers of the size of half a dollar or a dollar piece, dirty yellowish or pale yellowish, covered by a soft or firm cheese-like mass which can easily be removed. Such places become confluent and form irregular spots, the intestinal wall becomes thickened and loses in elasticity.

The mesenteric glands are always swollen. Acute swelling of the spleen, parenchymatous degeneration and inflammation of internal organs and occasionally the presence of metastatic abscesses indicate more or less that a general infection has taken place.

**Symptoms.** Gastro-enteritis sometimes begins with the symptoms of acute gastric catarrh and gradually passes over into the picture of inflammation; generally, however, the animals sicken suddenly under grave symptoms. The ingestion of feed and, in ruminants, rumination cease, thirst is sometimes much increased, often the animals exhibit manifestations of abdominal pains, which are generally dull, less frequently sharp and severe so that they lead to maniacal attacks. Carnivora and hogs vomit frequently, and the expelled matter occasionally is bloody; vomiting is sometimes seen in herbivora, and more frequently repeated retching and gagging.

All animals except the horse usually show tenderness on pressure of the region of the stomach or abomasum or even of the whole abdomen. In ruminants there is generally also moderate bloating with moderate increase of the circumference of the abdomen; this is, however, also in other animals, seen in the beginning of the disease. In the further course of the disease the dimensions of the abdomen usually decrease in consequence of diarrhea, and the abdomen is drawn in.

Whenever the stomach is affected particularly, the intestinal sounds are less frequent or entirely absent and there is constipation. If enteritis is already present the intestinal sounds are intensified or continuous and diarrhea sets in, the animals void large masses of fetid matter with painful efforts; these are sometimes mixed with shreds of tissue or blood; dogs sometimes void pure blood; relaxation of the rectal sphincter muscle finally leads to involuntary defecation. If diarrhea has existed for some time and if the intestinal contents have consequently been diminished materially, the intestinal sounds, depending both upon peristalsis and the presence of fluids and gases, may be absent; simultaneously with the absence of intestinal sounds



there occurs a decrease of the droppings and there may be complete constipation. However, tenesmus of the rectum is even then often observed.

In hemorrhagic gastro-enteritis of dogs loss of substance is seen frequently in the mucosa of the cheeks and on the gums, but this is rarely the case in dysentery of cattle.

The urine contains albumen, also occasionally formed elements such as casts, sometimes also blood, and it shows an acid reaction even in herbivora.

The symptoms described are accompanied by great prostration and grave general symptoms. An elevated temperature (Fig. 38) sets in from the start, but the height of the fever varies considerably in different cases. The elevation may occur gradually or the disease may set in with a high fever. In hemorrhagic gastro-enteritis of dogs the temperature soon goes down or it may be subnormal from the start. The pulse is accelerated from the beginning and it becomes small and soft. The heat distribution on the periphery is unequal, the extremities feel cold, the skin is covered with perspiration and later on the eyes sink in.

Finally the animals lie on the floor stupefied and motionless, or they show convulsive motions, dizziness, muscular contractions, or their behavior is like that of dumb staggers. Hemorrhages into the skin, into the mucosæ or into the retina are sometimes observed (Schindelka).

Croupous diphtheritic enteritis in the horse occurs in three different types. The most frequent type begins with great dullness and weakness of the patient, the gait is markedly staggering, the temperature rises to 40-41° C. or even higher, the conjunctivæ appear icteric, dirty discolored, injected. There is a lack of appetite, diarrhea sets in only after four to five days (see Fig. 38), the feces are at first mashy, then entirely fluid and fetid; they may contain pieces of pseudomembranes; before diarrhea has set in, dry feces may in rare cases be covered with shreds of croupous membranes. After diarrhea has appeared, a fatal issue generally soon occurs. The second type, according to Keilgaard, gen-

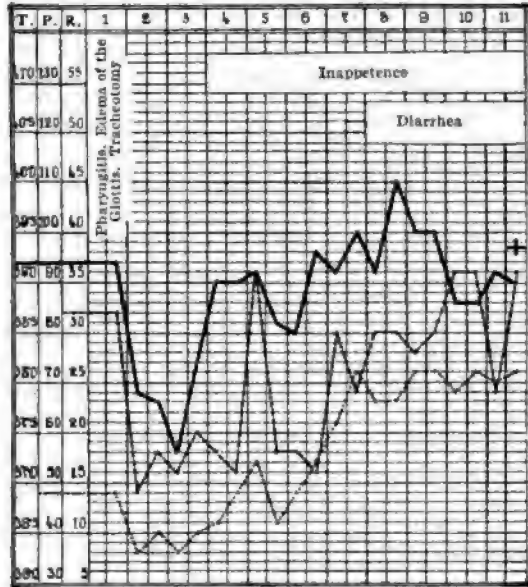


Fig. 38. Fever curve in diphtheritic enteritis in a horse.

erally commences with not very severe symptoms of diarrhea; elevation of temperature may be absent and a moderate elevation of temperature may be noted only on the last day of the disease. However, the pulse is accelerated from the start. The general condition of the animals is greatly disturbed and a marked emaciation soon occurs. During the slower course of the disease, pseudomembranes are shed more frequently than in the first type. The third type, according to Folger, commences with the symptoms of sleepy staggers; the dull animals show a staggering, waddling gait; they lean forward, press against the wall, but do not show any symptoms on the part of the intestines. The disease usually ends fatally.

**Course and Prognosis.** In very grave cases the disease usually terminates fatally within the first two days, even within the first twenty-four hours, while in ordinary cases it lasts from one to two weeks before death comes on from exhaustion, or a fatal issue may also occur after an apparent improvement. Recoveries are as a rule rare and convalescence is of very long duration.

The more rapidly the symptoms become worse, the less chance is there for recovery. Important unfavorable signs are a considerable acceleration of the pulse, profuse and bloody diarrhea or obstinate constipation. There are, however, cases, particularly among herbivora with a long intestinal canal, where there may be a mild course of gastro-intestinal catarrh extending over one to two weeks, which may suddenly become worse and end in death in one or two days. Enteritis in hogs, due to the bacillus necrophorus, not uncommonly takes a less virulent course.

**Diagnosis.** A generally sudden onset, a rapid deterioration with abdominal pains, obstinate diarrhea following constipation, weakness of pulse as a rule are characteristic enough to lead to a correct diagnosis and a differentiation from other milder and more local gastro-intestinal affections. However, since gastro-enteritis may be part of the picture of infectious diseases (see page 347), or of poisoning, one must in all cases be on the lookout for signs of the latter (on the skin, in the buccal cavity, in the respiratory tract, in the eyes), and one must also consider whether the affection appears sporadically or widely disseminated. In horses the possibility of intestinal tuberculosis must be considered; it also manifests itself by the symptoms of enteritis.

The cause of the affection cannot usually be recognized from the clinical picture but must be looked for in the history of the case, environmental circumstances, examination of the feed, etc. Secondary gastro-enteritis due to other disturbances of the stomach and intestines (accumulation of feces, coproliths, helminthiasis) is usually characterized by the fact that digestive

disturbances have for some time preceded the onset of grave enteric symptoms.

Fröhner gives the following **differential diagnosis** of disturbances due to various fungi:

1. Moulds ("Schimmelpilze," German). Lack of appetite, colic, constipation, diarrhea with bloody, slimy, occasionally very fetid feces, polyuria, dizziness, stupor, sleepy staggers, paralysis of the extremities and the tongue, amaurosis, profuse perspiration.

2. Blight-fungi (Ustilaginæ, "Brandpilze," German). Salivation, continued masticatory motions, tottering, staggering, general motor and sensory paralysis; in other cases, symptoms of gastritis.

3. Rust-fungi (Uredinæ, "Rostpilze," German). Dermatitis on the head (lips, cheeks, eyelids), conjunctivitis, urticaria, stomatitis, pharyngitis, glossitis, colic, bloody diarrhea, hematuria, paralysis, somnolence.

4. Yeasts (*Saccharomyces*, "Hefepilze," German). Intense cerebral excitement, followed by stupor and paralysis.

**Treatment.** If gastro-enteritis is due to the ingestion of spoiled feed, the early removal of the gastro-intestinal contents is the first indication. Emetics by the mouth or by subcutaneous injection may be used in hogs and in carnivora; but much better is lavage of the stomach (see page 306); in other animals efforts must be confined to the administration of mild laxatives such as castor oil, salts, calomel; (see page 331). Subcutaneous injections of drugs stimulating peristalsis, (eserine, arecoline, etc.) are less advisable, although they may be indicated in such cases where abdominal pains are absent. If there is reason to accuse noxious microorganisms introduced with the feed as the cause of the disturbance, one should attempt to prevent their multiplication in the intestinal tract by the administration of disinfectants such as resorcin, naphthalin, creolin, salicylic acid (see page 332). However, a good effect from these drugs can be expected only in gastritis, because they cannot accomplish much in the intestinal tract, and they may occasionally do more harm than good. Mucilaginous drugs and astringents (see page 332) may likewise be used advantageously. In hemorrhagic gastro-enteritis adrenalin or suprarenin in 0.1% solution, thirty drops every three hours for dogs (Uebele) are indicated as styptics. In cases of poisoning antidotes have to be administered; however, even in such cases washing out of the stomach and the administration of mild laxatives must be practiced.

If abdominal pains are severe they must be alleviated by opium or morphine (see page 332); prostration and stupor should be treated by cold douches and friction; however, douches must not be employed if the temperature is subnormal, when warm packs are indicated. A weak pulse calls for the administration of stimulants such as wine, whiskey, black coffee, tea internally; ether, oil of camphor, caffeine subcutaneously—in weakness and collapse. The infusion of warm physiologic salt solution into a vein or under the skin may be very beneficial, particularly if some adrenalin, suprarenin, or grape sugar has



been added (see page 333); such infusions may be repeated several times.

If the sick animals are still in a fair state of nutrition, they should be starved several days; the tormenting thirst must, however, be alleviated by lukewarm water. Dogs should also receive tea with some cognac. If there is great debility mucilaginous food should be given, such as soups of linseed, oatmeal, rice, sago with the addition of alcohol and eggs or wine, soups with the yellow of eggs, if the condition of the stomach permits feeding by mouth at all. If this is impossible, artificial feeding must be instituted (see page 333); this may be accomplished by rectal feeding or, if the rectum is likewise affected, by subcutaneous or perhaps by intravenous injections.

**Literature.** Albrecht, W. f. Tk., 1881, 1.—Bang, Maanedsskr., 1890, 235.—Berg, Maanedsskr., 1896, VIII, 236.—Berger, T. Z., 1905, 81.—Chaussé, Rec., 1905, 788.—Ficker, Z. f. Fhyg., 1906, XVI, 361 (Rev.).—Pröhner, Monh., 1892, III, 49.—Galtier, J. vét., 1887, 142.—Gerlach, Rinderpest, 1867, 61.—Glage, Monh., 1901, XIII, 550; XIV, 25.—Glässer, D. t. W., 1909, 513.—Heuss, Z. f. Vk., 1908, 201.—Keilgaard, Maanedsskr., 1907, XIX, 81.—Mohler & Buckley, Anim. ind., 1902, 297.—Müller, S. B., 1893, 21.—Oemler, A. f. Tk., 1882, VIII, 241.—Piorkowsky & Jess, B. t. W., 1901, 45.—Pusch, D. Z. f. Tm., 1893, XIX, 38.—Reynal, Diet., 1860, V, 149.—Schiel, B. t. W., 1906, 361.—Schindelka, O. Z. f. Vk., 1891, 90.—Szántó, A. L., 1908, 12.—Wedernikow, Vet. Jhb., 1893, 70.—Wrzosek, V. A., CLXXVIII, 82.—Wyssmann, Schw., A. 1907, XLIX, 129.

**Enteritis in Fowls.** Aside from secondary enteritis which occurs in the course of fowl cholera, hemorrhagic septicemia of chickens and other fowl-septicemias, in fowl diphtheria, in poisoning by acrid substances and finally in the presence of intestinal parasites (coccidia-enteritis, helminthiasis), primary croupous-diphtheritic processes are met with in the intestines of chickens, geese, ducks, turkeys and peacocks (Röll, Johne, Kitt, Guittard). Such affections may appear sporadically in animals of one species only or in animals of several species.

The **cause** of croupous-diphtheritic enteritis in fowl is not well known; however there is no doubt that one must look for infectious agents as their cause.

Fumagalli has seen an enzootic croupous enteritis in chickens caused by aspergillus.

**Post-mortem examination** shows, either in the small intestines (Röll, Kitt) or only in the cecum (Guittard), soft cylindrical croupous-diphtheritic masses as long as a finger, filling the intestines more or less completely; the mucosa is intensely reddened, swollen and hemorrhagic. In other cases the intestines contain a smeary-purulent, rice-waterlike or light reddish-gray discolored fluid; while the mucosa is either in a condition of serous infiltration and partial softening (Röll) or is covered by a veil of fibrin coagula, or it shows sharply contoured, blackish or greenish-gray places, elevated 3-4 mm. above the surface; still other evidences of inflammation may likewise be seen.

The **symptoms** of the disease are lack of appetite, depression, sluggish motion due to stiffness of the muscles, brown discoloration of the integument of the lower abdomen, violent diarrhea with yellowish droppings.

The disease usually takes a rapid course and the animals die often within a few hours (Röll) or after the disease has lasted from twenty-four to thirty-six hours; if the ceca alone are affected, death may follow in only seven to eight days.

The treatment is unpromising. Guittard recommends as a prophylactic the administration of a decoction of radix altheæ, and the addition of syrup, honey and naphthol to the feed; the latter should previously be softened in a weak solution of carbolic acid or salol.

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**Meat Poisoning and Botulism in Man.** Meat poisoning in man generally occurs after the eating of meat of cattle, calves, cows, sometimes also hogs and even horses which have become sick with septic or pyemic inflammatory processes (puerperal fever, purulent mastitis, purulent inflammation of serous membranes or joints, enteritis) and have been slaughtered while suffering from these affections. Particularly in summer, more or less extensive epidemics have been observed, variable as to the severity of the clinical picture and presenting sometimes solely the symptoms of uncomplicated gastro-intestinal catarrh, and at other times those of a gastro-enteritis or even typhoid affection with muscular weakness and ataxia. Frequently the picture is complicated by albuminuria, catarrhal pneumonia, circumscribed cutaneous erythema, urticaria or hemorrhages into the skin. The mortality is rarely more than 2-5%.

Bacteria concerned in meat poisoning of man may be divided into three main groups.

Type I. *Bacillus enteritis*: *Bacillus* of Frankenhause (Gärtner), B. of Moorseele (v. Emmergem), B. of Gent (v. Emmergem), B. of Brügge, Brussels, Willebroek (De Nobele), B. of Rumflett, Haustedt (Fischer), B. of Cotta (Neelsen, Johnes & Gärtner).

Type II. *Bacillus Aertryck* (belongs to the group of para-typhoid or of hog cholera bacilli): *Bacillus* of Gaustadt (Holst), B. of Breslau (Fluegge-Kaeuser), B. of Posen (Günther), B. of Hatton, Chadderton (Durham), B. of Sirault (Hermann & v. Emmergem), B. of Calm-phout (v. Emmergem), B. of Aertryck, Meirelbeck (De Nobele); to this group also belongs the *bacillus morhificans* Rosenau which occurs in septic processes in cattle and which, like the *bacillus enteritis*, causes purulent and necrotic foci in the liver and spleen of inoculated animals. Similar to the latter is the *bacillus* of Rotterdam of Pöls & Dhont.

Type III. *Bacterium coli*, *bacillus proteus*, etc. The bacteria of the third group get into the meat products after slaughtering and do not very often become the cause of meat poisoning if compared with the other two groups.

**Sausage poisoning** (botulism, allantiasis) has been noticed after the ingestion of sausage, corned or smoked beef, canned beef, conserved meat, venison, etc. Sometimes similar symptoms have been observed after the ingestion of salted fish (ichthyosism).

The cause of sausage poisoning are the toxic products (botulism toxin) of the anaerobic *bacillus botulinus* of Van Emmergem, which is found in meat preparations and taken with them into the gastro-intestinal tract of man. This toxin produces cloudy swelling and fatty degeneration in the cells of the parenchymatous organs and the lining endothelia of the blood vessels, also changes in the ganglion cells of the anterior roots of the spinal cord and in the bulbar nuclei.



Hence we observe in botulism symmetric motor paralysis, particularly in the region of the cerebral nerves (paralysis of accommodation, mydriasis, ptosis, aphonia, dysphagia), while gastro-intestinal disturbances are not infrequently absent or are only very insignificant. There is, however, obstinate constipation and suppression of urine.

The toxin of bacillus botulinus is not very resistant towards various reagents such as particularly alkalis; it is made innocuous when heated to 80° C. for one half hour.

Botulism is, therefore, an intoxication, while genuine meat poisoning is produced originally by pathogenic bacteria or by such that have become pathogenic; however the toxins of these bacteria, and ptomaines which have been formed, do likewise play a rôle in the disease-producing process.

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### 19. Bloating of the Intestines. Meteorismus Intestinalis.

(*Darmaufblähung*, *Windkolik* [German]; *Colica flatulenta*; *Indigestion intestinale gazeuse* [French].)

Bloating of the intestines or meteorism consists in an excessive dilatation of the intestines in consequence of the rapid formation of gas.

**Occurrence.** The prevalence of primary meteorism is intimately connected with the methods and conditions of feeding the animals. These conditions vary a good deal in different countries and parts of countries, hence the affection is not uniformly prevalent everywhere. In the Budapest Clinic it represents 12 to 15% of the colicky affections of horses; the lower percentages, however, are observed only rarely. Occasionally the affection occurs in hogs, dogs and rabbits. In ruminants the same causes lead to bloating of the rumen.

**Etiology.** Primary bloating of the intestines is due to the ingestion of bloating feed. Particularly dangerous are in this respect withered or heated fresh green feed (clover, alfalfa, esparsette, fresh grass), also beets and potatoes, rarely seeds of leguminosæ, more frequently crushed corn or barley. Mouldy feed may likewise be dangerous (Hendrick). Bloating in rabbits is not infrequently caused by fresh cabbage, kale, beets, raps, vetches, buckwheat. The ingestion of water in large amounts increases the danger from these foods materially, particularly leguminosæ and crushed grains may do harm under these conditions. Sometimes an unobjectionable feed, like oats and hay may cause bloating in horses if these animals feed greedily and



rapidly or are overworked shortly after feeding. Dogs are sometimes bloated after the abundant ingestion of starchy food. Abundant stuffing of parts of the intestines with thick mashy feces favors the collection of gases in the parts nearest to the stomach.

It sometimes happens that cribbling horses swallow enough air on feeding to produce bloating, and it is claimed that this may also occur if these animals move rapidly towards the wind. While the first mode of formation of moderate bloating may be conceded, the second one can hardly be accepted as probable.

**Secondary bloating** occurs in various forms of closure of the intestines in grave thrombotic-embolic disease of the intestine; and in the course of diffuse acute peritonitis, in internal strangulation, volvulus and thrombosis of the mesenteric vessels, bloating appears early after a few hours in the affected loops of intestines, while anteriorly to the obstruction, as in other cases of secondary bloating, this condition develops slowly and only rarely reaches a high degree during the life of the animal.

**Pathogenesis.** A portion of the feed swallowed by horses gets from the stomach into the small intestines even during feeding and from there soon into the large gut. According to Scheuner and Grimmer corn gets into the large intestines two hours after ingestion, hence fermentable feed can form gases, very shortly after ingestion, along the whole intestinal tract with the exception of the rectum and also to a moderate extent in the stomach. In consequence of stretching and by chemical influences (carbon-dioxide, methane, fatty acids) the muscularis of the stomach and intestines is irritated to frequently recurring convulsive contractions which in their turn produce colicky pains. The strong contractions and the absorption of the gases by the blood at first prevent great stretching of the intestines, but these means are insufficient to absorb all of the rapidly forming gas. Loops of intestines, therefore, become more and more dilated; these, as well as the whole intestinal tract, suffer in contractility while compression of blood vessels occurs likewise, further preventing the absorption of gases. The dilated intestines also press the diaphragm toward the thorax and the negative intrathoracic pressure is diminished, the cardiac diastole becomes interfered with and the blood pressure in the arteries is lowered.

In horses and dogs bloating is developed similarly though generally somewhat later after food ingestion.

**Anatomical Changes.** On postmortem examination the abdomen is occasionally dilated and very tense, the more so since fermentation and the formation of gases continue after death. The dilated and tense loops of intestines are pressed out with great force after opening up the abdominal cavity. If rupture

has occurred gastric and abdominal contents are found free in the abdominal cavity. The condition of the margins of the tea show whether the latter occurred during life or postmortem. In the former case the margins show bloody sugillation and possibly also swelling, and the muscularis may be retracted behind the mucosa. The thoracic organs show a high degree of passive congestion.

(The formation of gas, produced postmortem in not perfectly fresh cadavers of horses also causes a general dilatation of the intestines, signs of suffocation are however absent.)

**Symptoms.** In horses symptoms of primary bloating usually appears shortly after the ingestion of food and are similar to those of acute dilatation of the stomach (see page 299). As a rule the clinical picture is initiated by violent, frequently recurring attacks of colic. Since genuine colicky pains, due to convulsive contraction of the intestinal muscularis are not intensified by pressure, the patients throw themselves recklessly on the floor and roll about in a manner attracting attention; later on they sit down on their haunches (dog position).

Simultaneously with the symptoms of colic, the abdominal circumference increases rapidly, the space between the costal arches becomes greater, the abdominal wall protrudes in barrel shape; most marked is the increase of the abdomen in the flanks, particularly on the right side. Animals who even normally possess a tense and less yielding abdominal wall, will not show a very well marked enlargement of the abdomen, in spite of severe disease.

The percussion sounds are deep and resonant, more or less all over the entire abdomen, but particularly in the flanks; the sound becomes, however, somewhat higher and weaker in grave cases on account of the great tension of the intestines and of the abdominal wall. Sometimes the sound is metallic and not only over the cecum, where a similar sound is heard under normal conditions, but also over the other portions of the intestines.

The intestinal sounds are sometimes continuous at the beginning of the affection; in the further course of grave cases they become less frequent and may cease entirely; they are often high in pitch and metallic.

Rectal examination shows a high degree of dilatation of all accessible intestines with the exception of the rectum. Their walls feel tense and elastic. Bloating of the colon displaces its pelvic flexure deep down into the pelvis or towards the right side and in the latter case the tense longitudinal bands of the left lower portion run from the right towards the left and sometimes in a spiral arrangement. The left lower portion is now much dilated and reaches up to the left kidney; it is either beside or below it and the much thinner, but likewise bloated left upper portion of the colon is displaced by the raised lower



portion, either towards the median line or towards the left abdominal wall (Fig. 39).

Defecation at first occurs more frequently but soon becomes delayed or suppressed. In the beginning, and in the less severe cases until termination of the affection, there is a good deal of flatus.

The respiration becomes increasingly more difficult; the pulse rapidly becomes accelerated, so that when the bloating of the posterior abdomen has become quite noticeable the pulse will soon be 60 per minute; the more rapid it becomes the weaker it will be. The mucous membranes are at first dark red, soon, however, they become cyanotic, the visible veins are strongly filled.

The body of the patient is bathed in perspiration. If the stomach is likewise bloated there is also often belching, more rarely retching, occasionally vomiting.

Wöhner saw a case of bloating in a foal with subcutaneous emphysema on the back and shoulders. The case ended in recovery.

Secondary bloating leads to variable symptoms aside from the increase in abdominal circumference; these depend largely upon the nature of the underlying condition.

In hogs, dogs and rabbits, the clinical picture varies somewhat from that in the horse; the former animals either do not suffer abdominal pains or they only betray them by repeated attacks of crying out, frequent changes of position or groaning. Belching, retching and vomiting on the other hand are seen more frequently.

**Course.** The accumulation of gas may reach such a degree, occasionally within four to five hours, and in smaller animals earlier, even if the animals have apparently quieted down, that suffocation comes on. (Goldbeck saw a horse, in which bloating came on after eating fresh clover, succumb after the illness had lasted only 16 to 20 minutes.) Ruptures are sometimes seen; they occur in those cases of primary bloating where the accumulation of gas is not uniform and where some portions of the gastro-intestinal tract become excessively dilated.



Fig. 39. Bloating of the intestines. Position of the left loops of the colon and course of the longitudinal bands of the left lower portion in extensive bloating of the horse.



(Rupture is more frequently seen in the stomach. The diaphragm tears occasionally.) If rupture has occurred, the animals become suddenly quiet; however, the general condition deteriorates and collapse occurs rapidly (see page 301).

The affection not infrequently ends spontaneously in recovery; in the majority of cases recovery can, however, only be expected upon proper treatment. The course is always of short duration and usually it becomes obvious after twelve hours whether death will occur or whether improvement may be expected; if the latter is the case the symptoms of acute intestinal catarrh generally set in.

**Diagnosis.** The most important symptoms of primary intestinal meteorism are the following: rapidly increasing signs of colic after the ingestion of usually bloating feed, rapid increase in the size of the abdomen, loud sounds on percussion, and uniform, extensive dilatation and tension of all of the intestines (general intestinal meteorism). Simultaneously bloating of the stomach may be recognized by belching and vomiting.

Secondary bloating (see torsion or strangulation of the intestines; accumulation of feces, thrombosis of intestinal arteries, intestinal obturation, enteritis, peritonitis), which must be judged and treated differently, may be initiated with similar symptoms and must be excluded upon the basis of the history and upon the result of a rectal examination which should be made in every case of meteorism. Except in the case of peritonitis, secondary bloating is always confined to individual portions of the intestines (circumscribed, intestinal meteorism), and there are often symptoms which clearly point to the causative affection. Bloating occurring in consequence of an inflammatory condition is accompanied from the start by fever and the restlessness is not so marked. Torsion of the stomach in carnivora may be excluded when belching occurs and when the abdominal wall is not tender to pressure.

**Treatment.** In not too severe cases of primary meteorism one should attempt to stimulate intestinal contractions in order to remove the gases per vias naturales. Cold packs of the abdominal wall, cold douches to the latter, and cold water injected under some pressure into the rectum, favor the expulsion of gases in the horse by reflex irritation. One may also employ ether in water (15:400), salt solution, soap suds or much diluted turpentine. Massage of the abdomen or internal massage of the colon and cecum, applied with care through the rectum, are also beneficial; however, if the intestines are very tense one must not use this method, because rupture might be caused by it. Hummerich produced an extensive evacuation of the intestines by rolling the previously restrained horse on the back 15 to 20 times and then after an interval of time 8 to 10 times; this procedure may have to be repeated three or four times. Laxatives are also administered (salts with aloe, 150-200 gm. to 15-20

gm.); disinfectants are added to these to reduce fermentation (lysol or creolin, 15-20.0 gm. naphthol, naphthalin, 10-15 gm.). As long as the contractility of the intestinal loops is not abolished, as long as evacuation by it has not become impossible, and as long as intestinal gases are expelled from time to time, eserine (0.08-0.10 gm.) or arecoline (0.06-0.08 gm.) may be used subcutaneously. If, however, parts of the intestine have already lost their contractility (in very severe cases), these drugs are no longer beneficial and may be dangerous (see page 303). Since bloating of the stomach is frequently present simultaneously, the stomach tube should be used.

If, in spite of these applications, the condition of the patient becomes worse, or if the excessive dilatation of the abdomen bring about great dyspnea, puncture of the intestines must no longer be delayed. This removes the danger of suffocation and reestablishes contractility of the punctured portion of the intestines and the parts situated between the puncture and stomach. In secondary bloating intestinal puncture alone promises relief. Puncture of the cecum is usually practiced; if necessary, however, the colon or the small intestines must be punctured.

**Intestinal puncture** is best practiced with a slender trochar 10x15 cm. long. The skin is first properly cleansed, then an incision is made. The trochar is now introduced into the center of the depression of the right flank with the point directed towards the left elbow. After the escape of gas ceases, the tube is removed or, better still, left in place and closed with a cork, when it may be opened later on to let out more gas which might have again accumulated. If in spite of this puncture the symptoms of bloating persist or if the left flank has been distended more prominently from the start, the left flank is punctured. If this is done one must previously ascertain by rectal exploration at which point of the left side the bloated colon is situated, so that one does not puncture the rectum or some loops of small intestines or enter into the free abdominal cavity, in which case the puncture is without any result whatsoever.

Puncture of the colon from the rectum (Imminger, Föringer, Jensen, Reinhardt) appears indicated only in those rare cases, when the left portion of the colon is not lying close to the abdominal wall and when puncture of the cecum does not bring relief. Such a puncture may be made with a large exploratory trochar or an Elsässer trochar with a curved canula 40 cm. long or a Raitsits trochar which is short and can be introduced into the rectum as far as one can reach, and may in this manner reach anterior portions of the intestines. To permit the escape of the gases the short trochar must be connected with a rubber tube. If the free end of the tube is placed below water the evacuation of gas can be easily controlled.

Intestinal puncture, whether practiced from the flanks or from the rectum, is never harmful in primary bloating, if we use a slender trochar and proceed under aseptic precautions, or at least as cleanly as possible, and if in puncture from the rectum one follows the trochar with the guard. Before puncture from the rectum, the latter should be irrigated several times with disinfectant solutions. In secondary bloating, when the elasticity of the intestinal wall has suffered in consequence of serous or hemorrhagic extravasation, it may occur exceptionally that some intestinal contents get into the abdominal cavity.

Great restlessness must be counteracted by morphine injections (0.3-0.5 gm.) or by rectal injections of chloral hydrate, because reckless rolling may bring about rupture.

In hogs, dogs and rabbits, kneading or massage of the abdominal cavity may be beneficial; also chasing the patients around, also repeated cold douches. If the stomach is bloated simultaneously, emetics (see page 291) may be used. If suffoca-

of obstipation in horses, because feed of this type has to be taken in very much larger amounts than more nutritious food and it furnishes feces of much denser consistency, which are more difficult to move along. Such food stuffs are straw, particularly if cut into short chaff, or if it is taken up from the bedding straw (so-called straw feeders), corn stalks, hard-fibrous clover, alfalfa, etc. Constipation is, on the other hand, frequently produced by feeding materials rich in the salts of the earthy metals such as bran, crushed corn and barley, marshy hay, and also upon the ingestion of sand. All the above mentioned food stuffs act particularly unfavorably in sudden change of feed and with insufficient exercise.

Retarded movement of the large intestines may cause obstipation of the gut, even on proper feeding. This is seen in old, enfeebled horses, in fat animals which do not exercise much, usually also in horses of a listless temper. Insufficient peristaltic motion may also be consecutive to chronic intestinal catarrh, and the latter may have developed upon a thrombotic or embolic basis.

The cause of the disease is sometimes a disturbance of mastication (bad teeth), because then the feed is not sufficiently broken up before it gets into the gastro-intestinal tract. Insufficient peristalsis and frequent anomalies of teeth in old horses explain the frequency of obstipation in advanced years.

The combined use of morphine and atropine against shoulder lameness causes many cases of obstipation with subsequent bloating or rupture of the stomach, because atropine diminishes the intestinal secretion and morphine suppresses peristalsis.

**Secondary** obstipation is seen after intestinal stenosis, after intestinal obturation existing for some time, in combined paralysis of the tail and sphincter, and exceptionally following thrombo-embolic processes of intestinal vessels if these have lasted at least for several days.

**Pathogenesis.** Retardation of peristalsis and a firmer consistency of the intestinal contents bring about, even under normal circumstances, a gradual accumulation of feces, particularly in the narrower portions of the intestines, where the removal of the contents is slow, even under physiologic conditions. An accumulation of feces occurs most frequently in the stomach-like dilatation of the colon, in front of the first portion of the small colon and it may spread from here to other portions of the colon and even to the cecum. Sometimes obstipation develops in the pelvic flexure and at other times again in the cecum in front of the comparatively narrow opening of the colon; also fairly frequently in front of the ileo-cecal valve, rarely, however, exclusively in the rectum, in the region of the second flexure of the duodenum and only exceptionally in the jejunum.

The accumulation takes place in either one of two modes. The above mentioned portions of the small intestines, most fre-



quently the end piece of the ileum during or after one meal, rapidly become filled with coarse fibrous, dry feed (generally chaff). Since the gastric contents in horses enter the small intestines partly unchanged (Ellenberger), the feed mash, if insufficiently broken up or if in a rather dry condition meets an impediment at the ileo-cecal valve, occasionally even in front of second curvature of the duodenum or in front of any curvature of the jejunum. The development of obstipation in the large intestine which is much wider, occurs, however, much more slowly. The retardation of peristalsis, the firmer consistency of the intestinal contents, the greater mass of the intestinal contents, cause an increasing delay of their transport and they become more and more desiccated. The appetite of the animals has not suffered in the mean time, and larger and larger masses accumulate in the affected portions of the large intestines.

Sudden or rapidly occurring closure of the small intestines, with subsequent stretching of the intestinal wall by the accumulated masses of feces, stimulate the occluded portion and those anterior to it to convulsive contractions which produce colicky pains. The contractility of the muscularis of the large intestine, however, diminishes from the start only very gradually since the accumulation and the closure are brought about very gradually, in the course of several days. Convulsive contractions and colicky pains, therefore, do not occur or only very moderately, and they are generally localized at a point in front of the obstipation. The dilatation of the filled portion of intestines, however, produces a disagreeable feeling of tension or fullness in the abdomen, which in combination with the absorption of intestinal poisons causes an intoxication which in its turn produces dullness of the sensorium. An exception is presented by those rare cases where obstipation in combination with a change of feed will produce lively gas formation in front of the closed portion, especially in the cecum. Then the irritating gases will produce strong convulsive contractions of the muscularis.

According to whether the transportation of accumulated feces is stopped suddenly or within a short time, or whether as in obstipation of the large intestines, the cessation occurs very gradually, contractions of intestines behind the obstructed portion cease after a few hours or only after several days, then the periodical filling of the rectum likewise ceases. An exception to this rule sometimes occurs in obstipation of the cecum in those cases where the desiccated fecal masses do not reach up to the cecal-colonic juncture and where the contents of the head of the cecum can be pressed partially into the colon.

The dried masses of feces may subsequently cause necrosis of the epithelial covering of the mucosa, and may in this manner lead to enteritis, or even to rupture of the wall of the gut. The decomposition of the thin-fluid feces accumulating in front of the point of obstruction in the course of time

causes moderate bloating, possibly even enteritis; since there is no fermentation in the obstructed portions of the intestines, bloating will not occur there. However, in obstipation of the small intestine and not infrequently in obstipation of the large intestine, secondary dilatation of the stomach is seen frequently. Enteritis and the other complications cause general symptoms, which, however, come on only after several days in obstipation of the large intestines.

**Anatomical Changes.** The obstructed parts of intestines are dilated, their surface may be smooth or nodular, their abundant contents appear more or less desiccated, mortar-like; the contents of the large intestines may even show the contours of the pouches. The mucosa shows blood extravasation; it looks as if covered with bran in consequence of epithelial necrosis, and sometimes shows larger patches of necrotic tissue. In more prolonged obstipation of the cecum one sees hypertrophy of the muscularis and considerable chronic dilatation of the intestines. Sometimes a rupture is found at the place of the obstruction or immediately in front of it; if there has been dilatation of the stomach there may be rupture of the stomach.

**Symptoms.** In the most common type of **obstipation of the large intestine** one sees in the beginning retarded defecation which may last for several days, then there is complete absence of it in spite of repeated efforts. Obstipation of the cecum offers an exception because there may be defecation, though perhaps deficient, which only ceases in the further course of the affection. With the absence of defecation symptoms of colic appear, at first of a mild type and at long intervals, often lasting for hours. Restlessness later on becomes somewhat more continuous and intense, but does not reach a high degree. The animals lie fairly quietly on the floor, only occasionally turning their heads toward the abdomen (rolling is rarely observed); they paw from time to time with their front feet and move their tails. Squatting on the haunches may occur in any form of obstipation. Not infrequently the patients place their feet like male horses in urination, so that the front legs are placed much towards the front and the hind legs much backwards, while the back is stretched out.

The abdominal circumference remains unchanged for some time, and occasionally until the disease has run its course, particularly if the horse has a tense abdominal wall. The percussion sound is usually dull over the colon, and the dullness may extend over the cecum; often, however, percussion may not reveal anything abnormal in spite of extensive accumulation of feces, because the loops of intestines filled with firm fecal masses may not be in touch with the abdominal wall. The intestinal sounds always occur less frequently and they may be suppressed entirely in the further course of the disease.



Rectal examination reveals dilated, semi-solid, soft or entirely firm loops of intestines. If there is an accumulation of feces in the colon, which is quite frequent, one finds the whole left half of the abdominal cavity filled with the left portions of the colon, the lower one being characterized by longitudinal bands and pockets and being felt towards the median line, the smooth upper portion becoming larger towards the thorax, and the two other portions in the entrance of the pelvis leading to the smooth pelvic flexure. The latter frequently becomes displaced entirely into the pelvis or slides towards the right side. If this is the case one can feel the longitudinal bands running from left to right, possibly with a spiral twist. Not infrequently the left upper portion of the colon is displaced to the right or to the left, beside or even below the dilated lower portion, but without the production of a true torsion. The enlarged stomach-like dilatation of the rectum may also be palpated in horses unless they are large; it can be felt in front of the cecum, a little to the right of the median line, and it has the shape of a large semi-spherical, tough body, moving synchronously with the respiration, possibly covered by the anterior mesenteric root. The beginning of the small colon may be felt under the anterior pole of the left kidney, as a sausage-like body of the thickness of an arm, running obliquely from right to left. The cecum, filled with feces, is detected when the hand is directed to the right flank; it is recognized by its form and by the course of its two longitudinal bands; it may be as firm as the colon under similar conditions or its contents may even be almost as hard as a rock. The head of the cecum and the small intestines are frequently bloated in cecal obstipation. Loops of the small colon in which feces have accumulated are felt, by the hand introduced into the rectum as sausage-like loops, at the entrance of the pelvis and particularly to the left, externally or internally to the left portion of the large colon; they are provided with one longitudinal band and filled with balls of fecal matter.

The pulse and respiration remain normal for a long time, even for days, or if abnormal at all they present only slight changes from the normal. The appetite may be good in the beginning of the affection or for several days, even after the first symptoms of colic, the animals ingest some feed and this aggravates their condition and the restlessness after each meal. Later, however, the appetite becomes permanently abolished.

In a more advanced stage, usually only after several days, there is elevation of temperature, with debility and accelerated pulse, indicating the advent of complications (enteritis, peritonitis, meteorism, dilatation of the stomach).

E. Bauer has made interesting observations concerning indican in the urine in obstipation. In all cases examined an increase of indican was found; in obstruction of the cecum three to four times the normal amount, while in obstruction of the colon a rather moderate increase was usually found. If the accumulation of feces could be removed from



the entire intestinal tract, the amount of indican became diminished below normal, but it remained high if the cecum could not be evacuated, even if diarrhea had been produced.

The symptoms of **obstipation in the small intestines** vary from those in obstipation of the large intestines since they come on suddenly, within a few hours after food ingestion, or directly after the latter, if the duodenum is involved. The symptoms of colic are decidedly more marked, sometimes quite severe, and a stretched position of the animals, as in urinating of male horses, is observed. Defecation ceases within a few hours. Rectal examination reveals the extended ileum as a smooth cylindrical mass the size of an arm, situated in the plane of the posterior pole of the left kidney and to the right of the vertebral column running from above obliquely downward and backward and to the right, or on the contrary from below on the left upwards and to the right towards the base of the cecum and continuous with the latter. The obstructed duodenum can likewise be felt as a cylindrical smooth body, the size of an arm, extending immediately behind the anterior root of the mesentery in a curved direction from the right to the left and adherent to the mesentery only by a short band.

In contradistinction to what happens in obstipation of the large gut, acceleration of the pulse and respiration appear one-half day or sooner after the occurrence of obstipation of the small intestine. This is obviously due to a secondary dilatation of the stomach or to early inflammatory changes of the mucosa, the pulse in particular rising to sixty and more per minute even on the first day of the disease. The appetite is completely suppressed.

**Complications** are not at all rare in either form, but are more common in obstipation of the small intestine. In the latter, that is, in one-third to one-half of the cases, secondary dilatation of the stomach (see page 297) occurs, although this condition is not at all rare in obstipation of the large intestine. Dilatation of the stomach may lead to rupture of the stomach. Rupture of the intestines, which is tolerably common in obstinate and unyielding intestinal obstipation and which always occurs in very extensive fecal accumulation in the cecum, like rupture of the stomach, leads to collapse (see page 301), or if the tear is not very large and the shock has not been too great, it will subsequently be followed by general acute peritonitis. A not infrequent complication in obstipation of the small intestines is enteritis.

**Course.** Obstipation of the large gut develops, as stated, very gradually; restlessness sets in only after a few days and the condition becomes worse by and by. The patients, however, are more or less dull during the whole course of the disease. Cases are seen occasionally where, after several days, the dis-

ease ends in recovery without having led to symptoms of colic. Fecal accumulation in the small intestine causes sudden attacks of colic and usually lasts one to two days. Obstipation of the large intestine always lasts several days, even two to three weeks (especially obstipation of the cecum), unless it is relieved earlier by proper treatment.

A recurrence of the attacks of colic is not infrequently observed, either because errors of diet have not been corrected or because the intestinal muscularis has been weakened while the obstipation lasted, or because the accumulated feces have not been removed completely. The two latter circumstances are particularly effective on account of anatomical conditions in obstipation of the cecum; and this form of fecal accumulation is seen not infrequently in the form of recurring attacks (so-called habitual chronic or periodic colic).

**Diagnosis.** Retardation followed by cessation of defecation, the history of the case, and the usually mild attacks of colic occurring at long intervals, the absence of general symptoms make a diagnosis of obstipation in the large intestine quite probable; however, only rectal examination can make the diagnosis absolute; its differentiation from similar affections, and its exact localization depend upon rectal exploration. Feces containing sand point to obstipation due to this (so-called sand colic), and in this type the general symptoms usually come on sooner. Valuable assistance in the diagnosis and prognosis may be obtained by the quantitative determination of indican as proposed by Bauer. It is absolutely impossible to determine the seat of the obstipation from the behavior of the animals as suggested by Klemm.

The size and the firm consistency of the affected parts of the intestines, which can be ascertained by rectal examination, distinguish the disease from various other forms of intestinal occlusion, with the exception of stenosis or obturation of long duration, or of paralysis of the rectum; these can be differentiated by the different nature of their onset and by the fact that the site of the stenosis or of the obturating foreign body can be felt from the rectum. In affections of the stomach and in obstruction of the small intestine, the contents of the large intestines likewise become desiccated if the disease has lasted for any length of time; however, the large intestines are not dilated by the desiccated feces but are diminished in diameter.

**Prognosis.** If proper treatment is instituted in good time, the great majority of cases end in recovery. The longer the disease has lasted the firmer the stagnating intestinal contents are, and the more dilated the obstipated intestines the less hope there is to remove the obstruction. One also must always consider the exact site of the obstipation, because if it is in the small intestines or cecum, a fatal issue is comparatively fre-

quent, and recurrences often occur. In obstipation of the cecum the reappearance of defecation does not always indicate improvement, because masses of desiccated feces may remain behind in the cecum. If complications have already occurred there is no hope for recovery, except in secondary dilatation of the stomach, which can be treated successfully.

In the Budapest Clinic the mortality for different years ranges between 3 to 10%.

**Treatment.** To remove accumulated masses of feces from the large gut, those which can be reached manually from the rectum should be evacuated, then a rectal injection of a large amount (30-40 qts.) of lukewarm water should be given, being allowed to run in under a low pressure. The injected water is partly soon expelled, but Dammann and Marek have shown experimentally that in horses without obstipation the fluid may travel up to the middle or even to the beginning of the colon, and that it may therefore be expected to produce softening of the feces in obstipation. Cold water is not well adapted for this purpose because it stimulates the intestines to contract and to prevent the progress of the fluid inward. The injection of a few quarts is of no avail, because the fluid will then irrigate only the posterior portion of the rectum. Accumulations of feces in the cecum or in the small intestines cannot be influenced even by very large quantities of water. The injections (which are not usually successful by themselves) must be repeated several times.

In order to soften the masses of feces which are situated more towards the stomach and to stimulate peristalsis, neutral salts in large doses should be administered (250-500 gm.), or aloes (30-40 gm.), according to Höhne, even 50 gm., or castor oil (300-500.0 gm.) with one-half to one quart of neutral oil, or with 50-70 gm. of ether. In cases which are not very grave, abundant defecation and speedy recovery are usually observed after the administration of these drugs. In grave cases, however, the rectal injections and the administrations of the above drugs must be supported by medicines which stimulate the intestinal muscularis to strong contractions. Not earlier than half a day after the rectal injection, or after the salts or castor oil have been given (in obstipation of the ileum, however, after a few hours), one should administer subcutaneously eserine (0.06-0.08 gm.), with pilocarpine (0.15-0.25 gm.) or with arecoline (0.05-0.08 gm.). These applications do not only stimulate the intestinal muscularis but they increase the intestinal secretions and make the mucosa more slippery for the expulsion of the masses of feces. Without such preliminary softening, the above named drastic means may produce rupture; this might particularly occur if eserine alone is used in somewhat large doses. It is necessary to watch the animals carefully after the use of the above drastics because they intensify abdominal pains, and the horses must be prevented from reckless throwing and roll-



ing in order to prevent the occurrence of intestinal rupture. It is usually necessary to repeat the administration of the above drastic medicines. In obstipation of the cecum Höhne repeatedly gives 50 gm. of aloes at intervals of three days, the treatment to be continued, if necessary, for two weeks; good results are claimed for this treatment.

The effect of different forms of treatment is assisted by massage or kneading of the masses of accumulated feces from the rectum (Sobelsohn), this further causes the intestines to contract and the fecal masses which can be reached may be broken up or moulded. Massage alone is usually not successful, except in obstipation of the small intestines if the jejunum can easily be reached by the hand introduced into the rectum. Hummerich and Kalcher obtained good results in obstipation of the large intestine by rolling the animals on their backs (see page 362).

In very severe obstipation of the ileum which does not yield to any of the forms of treatment described, one might try laparotomy to remove the desiccated contents through the left sided laparotomy wound, either by propelling them by hand or by taking them out after enterotomy. In obstipation of the large intestine, laparotomy does not appear indicated, because one cannot remove the great masses of desiccated feces completely and the sutures would not keep after partial removal, being torn by the masses which have remained behind. The cases of Gaullet and Hobday prove conclusively the danger of laparotomy in obstipation of the large intestine. Deghilaige has massaged intestinal loops through an incision made into the upper vaginal wall of a mare and has in this manner brought about recovery.

Secondary dilatation of the stomach can only be treated by the use of the stomach tube (see page 304). This instrument should always be used in obstipation of the small intestines, even before symptoms indicate dilatation of the stomach. If the gastric tube is used repeatedly under these conditions, rupture of the stomach can usually be prevented.

Obstipation in the large intestine also calls for dietetic treatment. Instead of rough feed or grain, which horses with obstipation of the large intestine take by preference for some time or after a temporary improvement, but which will surely lead to an aggravation of the affection, the animals should receive juicy food stuffs, like bran or flour gruels, beets, bulbous plants, green soft feed, always in small amounts and at intervals. The animals should be prevented from eating bedding straw by the use of a muzzle or otherwise. Pasturing the animals is very good. However, they should not be fed on any bloating green feed, particularly not in obstipation of the cecum.

**Prophylaxis.** Animals inclined to suffer from obstipation should be fed with fresh hay and green feed or molasses, or they should be pastured at certain times; if this cannot be carried out, the systematic administration of salts with the feed may properly be substituted to a certain degree. Faulty teeth must be repaired. The predisposed animals should be subjected to close observation, so that one can take the proper steps whenever there is any disturbance in defecation. Ducasse recom-

tends in such cases the injection of pilocarpine followed by eserine.

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#### (b) Obstipation in the Intestines of Carnivora.

**Occurrence.** Accumulation of feces in the intestines is common in dogs, much rarer in cats. Fröhner, in 70,000 sick dogs during the years 1886-1894, saw obstipation in 2% of the patients. In the Budapest Clinic 2 to 3% of the dogs had obstipation; in 1903, exceptionally, 7%.

**Etiology.** Obstipation is frequently produced by hard bodies, especially fragments of bone, more rarely fruit stones, pebbles or portions of soil which interfere with the passage of the feces. Dry feeding, exclusive or abundant vegetable feed (bread, dog biscuits, leguminosæ, flour paste) or exclusive feeding with bones, produce dry, mortar-like feces which can be moved along only slowly. Hairs, blades of grass, etc., which form firm masses in the feces act similarly. Obstipation, due to swallowed hair, occurs not infrequently in cats.

Retarded peristalsis is the cause of obstipation when it occurs in consequence of lack of exercise (chained dogs, house dogs), or when it is seen in older, debilitated animals. Chronic intestinal catarrh may also lead to insufficient peristalsis.

The disease occurs as a secondary affection in consequence of stenosis or obturation of the intestine, of painful conditions of the abdominal muscles (rheumatism, pachymeningitis), or of the neighborhood of the anus (inflammation of the anal glands), or after matting of the hairs around the anus. Obstipation is also usually present in chronic diseases of the cord.

**Anatomical Changes.** Feces collecting in some portion of the intestines, usually in the colon or rectum, form very dry, mortar-like, dark brown lumps. There are cases in which such lumps of feces form firm cylinders as thick as an arm, which fill the whole of the large intestine (Kitt); exceptionally the whole intestinal tract may be filled with similar masses of feces (Fröhner). If the affection lasts longer, necrosis, hemorrhagic or diphtheritic inflammation and even perforation of the intestinal wall may develop.

**Symptoms.** In spite of repeated efforts the animals cannot defecate or they void small dry lumps of feces, the surface



of which is occasionally covered abundantly with mucus or blood; this occurs when the mucosa has become inflamed or has been injured. Sometimes, in spite of existing obstipation, the animals void a thin-fluid, very fetid stool; this occurs when the obstipation mass has become softened at the periphery or in the center, so that the fluid contents from portions of the intestines nearer to the stomach can pass by.

The abdomen is sometimes drawn in, sometimes bloated, and the abdominal wall is then tense. On palpation of the abdomen one feels in front of the pelvic inlet, below the vertebral column, and parallel to it a cylindrical, firm, sometimes hard mass, variable in length (so-called fecal tumor, fecal cord). The mass may be felt behind the liver and sometimes even further up on the right side of the abdominal cavity and it is freely movable; this mass is formed by the dry fecal masses in the rectum and large intestines. In some cases, however, only one or several, occasionally very large lumps of feces are found.

Sometimes swelling and reddening of the neighborhood of the anus can be observed. The finger introduced into the rectum feels hard fecal masses; sometimes also fragments of bone; if a rectal speculum is used one sees the lumps of feces, also a dark red discoloration of the mucosa, which may be covered by gray membranous deposits.

The behavior of the animals varies from case to case. The repeated fruitless efforts cause some anxiety and the irritation of the intestinal wall occasionally produces pain expressed by whining and curling up. In other cases the animals, in spite of obstipation which may have existed for days, are comparatively quiet and perhaps only show some listlessness and sluggishness. Their gait is stiff, they hold the tail straight or strongly curved at its root.

The appetite may be preserved during the first day and this aggravates the condition. The appetite diminishes, however, in the further course, and finally disappears while the thirst becomes increased. Vomiting occurs exceptionally and particles of fecal matter may be expelled.

The temperature remains normal for days, but if enteritis has followed upon obstipation the temperature becomes elevated and septicemic fever may finally set in.

**Course.** Obstipation of a not too severe type may be overcome by the animals' own repeated efforts and, provided that they have not been too long neglected, even grave cases, will end in recovery if the proper treatment is instituted. If the disease is left to itself after it has arrived at a later stage, there will be progressive deterioration until enteritis, septicemia, peritonitis or occasionally uremia (in consequence of compression of the first portion of the urethra), close the clinical picture. If the morbid affection has lasted a longer time, i. e., two to three weeks or longer, even the proper treatment cannot save the



animal any more, on account of the gangrene of the intestinal mucosa which has occurred.

**Diagnosis.** Constipation in spite of fruitless efforts, combined with the detection of desiccated fecal masses in the rectum or colon, point to the correct diagnosis. If the animal has previously been well and if the accumulation of feces can be explained from the character of removed fecal masses, one may assume primary obstipation. Otherwise a careful examination of the organs is necessary in order to determine whether we are not dealing with a case of secondary obstipation. Chronic diseases of the spinal cord (including its membranes) have to be considered particularly, because these often first attract our attention through an obstinate obstipation. Diseases of the region of the anus and of the pelvic organs can be recognized easily on the basis of the history and as the result of palpation (sometimes to be carried out during narcosis). After long continued starvation or preceding diarrhea, defecation may be infrequent, but in such cases there is no accumulation of feces.

**Treatment.** If obstipation has not lasted long and if the lumps of feces which can be felt from the rectum are not very hard, the local treatment may be confined to the introduction of large masses of water into the rectum; this softens the lumps of feces, makes them slippery and stimulates the rectum to contract. One may use pure lukewarm water, better soap suds or water with oil, or pure oil. Thin fluids are allowed to run into the rectum from an irrigator or a funnel provided with a rubber tube; low pressure must be used. The softening of the feces may be hastened by cautious pressing and kneading. This simple procedure, which must eventually be repeated, usually suffices to remove the morbid condition. In mild cases glycerin enemata are sometimes sufficient.

When the obstipation has lasted for a considerable time, and when mortar-like masses or fragments of bone are present in the rectum, they should be removed with the finger or with a pair of forceps; then the more anteriorly situated, usually less hard masses, can be softened and removed with lukewarm water irrigations; in such advanced cases massage is not indicated. The artificial removal of fecal masses and rectal irrigations sometimes have to be repeated for several days, until it has finally been possible to remove piece-meal a fecal cylinder which may have been 20-30 cm. long.

Laxatives should be used only in recent cases or after the mechanical removal of the fecal masses near the anus, because if used in old cases by themselves alone, they may make the condition worse. The prescriptions to be employed are: Castor oil (1-3 tablespoonsful in an emulsion with 1-3 to 1-4 part gummi arabicum and 5 parts of water, or in gelatine capsules 3-5 gm. each), calomel (dogs 0.20-0.30 gm., cats 0.1-0.15 gm.), aqua laxa-

tiva viennensis (50.0-100.0 gm.), occasionally with the addition of sodium and potassium tartrate (5-10 gm.), extractum cascaræ sagradæ (2-10 gm.), phenolphthalein (10 gm. for large, 5 gm. for small dogs). The neutral salts (see page 332) may likewise be used, and in very obstinate cases drastics may be employed (1-5 drops of croton oil in 10-30 gm. of castor oil); also tubera jalapæ (0.2-0.4 gm.). Drastics must be reserved for those cases where no inflammation of the intestinal wall is present and where fragments of bone or other hard bodies have not penetrated into the wall of the gut.

Where accumulation of feces is due to a tumor, an abscess, an enlarged prostate, a cicatric, surgical interference is necessary.

The diet must be regulated so that the patients do not receive any food containing flour (bread, dog biscuits, potatoes, vegetables) or bones, but exclusively fluid food, such as broth, soup, milk. Animals which are predisposed to obstipation should receive as little of carbohydrates as possible, and they should be exercised frequently.

In order to prevent obstipation due to swallowed hair in cats, Grobon recommends (Rev. vét. 1906, 21) to brush the animals daily and to administer castor oil twice a month.

### (c) Impaction in the Intestines of Ruminants.

**Occurrence.** Double mastication, preliminary preparation and mixing with abundant fluid in the fore-stomachs of ruminants make primary fecal impaction of rare occurrence in these animals. The disease has been seen in cattle and in goats.

**Etiology.** The occurrence of primary impaction in ruminants generally depends upon continuous feeding with undigestible, dry feed (leaves or matted hay). Moist mouldy straw, withered dry and rotten cabbage may also be the cause of the disease. Mathis saw grave cases of fecal impaction in cattle after long railroad transportation.

**Symptoms.** The disease is manifested by obstinate constipation, by moderate bloating after each meal, however without the accumulation of feed masses in the rumen, gradual decrease of appetite and retardation of rumination. Signs of restlessness are either lacking or are very insignificant. The feces are dry like peat, sometimes covered with a good deal of mucus and are voided in small amounts in spite of efforts. The intestinal sounds are absent or weak. Rectal exploration reveals, in the region of the right flank, sausage-like loops of intestines filled with firm feces; in small ruminants the desiccated feces may be felt through the abdominal wall.

The affection ends fatally only in the very gravest cases, otherwise it usually leads to recovery within one week.

**Diagnosis.** Displacements of the intestines can be distinguished from impaction by the severe symptoms of colic and by a rapid deterioration. Stenosis of the intestines can often be differentiated only by rectal exploration.

**Treatment.** Injections of large quantities of water and the administration of neutral salts (500-1000 gm. for cattle, 50-100 gm. for goats) are indicated; also aloes (40-60 gm. for cattle, 10-20.0 gm. for goats); tartar emetic (10-20.0 gm. for cattle, 0.2-2.0 for goats) alone or in combination with salts (tartar stib. 15.0 gm. sod. sulph. and magn. sulph. aa. 500 gm., given in three doses during one day for cattle). Eserine, eseridine, pilocarpine or arecoline may likewise be indicated (see page 254).

The diet should be made up of potatoes, beets, bran or flour soups, green feed, leaves of beets.

**Literature.** Dieckerhoff, *Spez. Pathol.*, 1892, II, 450.—Eber, S. B., 1896, 30.—Mathis, J. vét., 1897, 459.—Röbert, S. B., 1893, 120.—Rychner, *Bujatrik*, 1841, 113.

#### (d) Impaction in the Intestines of the Hog.

**Etiology.** Hogs develop impaction of the bowel in consequence of exclusive dry feeding with grains or after ingesting short cut chaff, or after the ingestion of much sand if the latter has not caused enteritis. Sometimes continued stabling causes the affection.

Secondary impaction is frequently seen after chronic hog cholera or chronic tuberculosis, or after stenosis, due to a slowly progressing enteritis.

**Symptoms.** One observes diminished appetite, increased thirst, frequent grunting and efforts at defecation. In the beginning the animals are still able to press out some lumps of fecal matter, later on the constipation becomes complete. On palpation of the abdomen of not too fat hogs, one can feel large intestines filled with desiccated feces; occasionally one can detect adhesions of loops of intestines. Oppenheim saw in a hog with a high degree of impaction, retention of the urine, due to compression of the neck of the bladder.

Grave cases end fatally in consequence of enteritis.

**Treatment.** Abundant injections of water (2-4 qts.), manual or instrumental removal of the fecal masses accumulated in the rectum, followed by the use of laxatives, usually relieve the obstipation. Neutral salts (20-50.0 gm.) are best added to the drinking water or powdered upon the tongue of the animal. Since constant squealing of the hogs during the administration of the laxatives may give rise to aspiration into the lungs, other



cathartics should be introduced into the stomach by the aid of the stomach tube (tartar emetic 0.5-1.0 gm., castor oil 50-100 gm., dissolved in water or mixed with it), or eserine (0.005-0.02 gm.) given subcutaneously. Tartar emetic, castor oil or calomel may be given in the shape of an electuary. Dietetic treatment consists in feeding bulbs, fallen fruits, green feed, cut pumpkin, sour milk or whey.

**Literature.** Oppenheim. T. Z., 1909, 227.

#### (e) Impaction in the Intestines of Rabbits.

**Etiology.** Rabbits develop impaction of the large intestine after too abundant or exclusive dry feeding, particularly after dry bran or undigestible food stuffs have been given continuously.

Secondary impaction occurs after chronic intestinal catarrh in the presence of enteroliths and sometimes in consequence of painful affections of the rectum or of the parts in the region of the anus.

**Symptoms.** There is at first retarded defecation, then complete constipation, diminution of appetite and listlessness. Palpation of the abdomen, which is very easy, reveals the presence of desiccated masses of feces in the large intestines.

If proper treatment is instituted early the disease ends in recovery.

**Treatment.** Repeated rectal injections of water, and soap suppositories are to be employed (from tea to table spoonful doses). Syrup with milk in equal parts, pills of the leaves of senna (1-2.0 gm.) or of rheum (0.2-0.4 gm.). Tincture of rheum (5-10 drops in water), also root of jalap (0.2-0.3 gm.), or calomel (0.05-0.2 gm.) may likewise be used.

The diet should consist in green feed, juicy roots, lettuce, etc.

**Literature.** Braun, Kaninchenkrankheiten, 1907, 109.

#### (f) Impaction in the Intestines of Fowls.

**Etiology.** The disease is rare in domestic fowls which are kept in the open air; if it occurs at all it is generally due to foreign bodies (pebbles, feathers, animal parasites, pieces of rags). More frequently, although still rarely, the disease is seen in over-fed, highly bred fowls kept in confinement in cages, or in house birds. These animals suffer from a combination of heavy undigestible feed and sluggish peristalsis. Impaction in the cecum, due to improper feeding, appears in enzootic form, particularly among young turkeys. The matting together of

the feathers around the cloacal opening may likewise prevent defecation.

**Symptoms.** The birds drop dry feces in small amounts at long intervals and with effort; this attracts attention since under normal conditions their feces are dropped with great ease and are soft and mushy in consequence of the admixture with urine. If obstipation has lasted for some time there is listlessness, depression, lack of appetite, and attention is attracted to the birds even if the irregularity in defecation has escaped notice.

If the disease is neglected it may lead to death in consequence of necrosis of the intestinal wall, and exhaustion.

**Treatment.** If there is a mechanical impediment it must at once be removed; matted feathers may be untangled after wetting them with water; desiccated masses accumulated in the cloaca can be removed with a spoon. To remove the feces contained in the rectum, the careful injection of olive oil or soap water is to be recommended; in small birds a dull sound dipped in glycerin or castor oil may be introduced into the rectum.

If the impaction is in some part of the intestines nearer to the stomach, castor oil (table or tea spoonful doses), rheum (0.4-0.6 gm. with butter or honey in pill form), or eventually calomel (0.05-0.2 gm.) may be used with advantage. Neutral salts may be given in weak solutions (1:200) with the drinking water.

The dietetic treatment requires green feed, soaked grains, boiled bran, lettuce, carrots, fruit.

## 21. Internal Closure of the Intestine. *Obturbatio intestini.*

By obturbation of the intestines is meant a sudden narrowing or a complete closure of the lumen of the intestine by some body situated in the bowel. The affection occurs almost exclusively among horses and carnivora.

**Occurrence.** Obturbation of the intestines is a rare affection of horses. Among the horses of the Prussian Army this form of obstruction of the intestines was seen in seventeen years only in 0.3% of the cases of colic; in the Budapest Clinic it formed 0.2 to 0.7% of the cases of colic. (Holterbach claims that foreign bodies in the duodenum are not rare in cattle.)

**Etiology.** Obturbation of the intestines in horses is usually brought about by enteroliths or lumps of fecal matter, more rarely by parasites, exceptionally only by foreign bodies.

Intestinal calculi (enteroliths) are mainly composed of phosphate of ammonia and magnesia (90%); they also contain some ( $\frac{1}{2}$ -1 $\frac{1}{2}$ %) carbonate and phosphate of lime, common salt,

and chloride of calcium (Fürstenberg). The most frequent cause of calculi formation in the intestines is the continued abundant feeding of wheat and rye bran (the disease is frequently found among the horses of millers and bakers), which contain an abundance of phosphate of magnesia. The latter dissolves in the acid contents of the stomach and of the small intestines, and is precipitated in the alkaline contents of the large bowel; it combines with the ammonia formed during putrefactive processes, and gives rise to phosphate of ammonia and magnesia, crystallizing around bodies which may have accidentally gotten into the large intestine (grains of oat, pebbles, splinters of iron, swallowed tooth [Fobelot]). Zschokke, however, claims that intestinal disturbances play a considerable rôle in the production of intestinal calculi. The latter are formed in the large intestine, as a rule preferably in the stomach-like dilatation of the colon. We here usually find one calculus, variable in size, generally more or less spherical, sometimes weighing over twenty pounds; there may exceptionally be several calculi, even many of them; in the latter case they are irregularly formed. Colin examined horses for calculi; he found them 23 times (2.5%) in 900 cases examined, always in the stomach-like dilatation of the colon, only once in the cecum. Calculi have exceptionally been found in the small intestine; their mode of origin could then not be explained; in some of these cases the calculi were probably gallstones, as pointed out by Lewin.

According to Fürstenberg a new layer is formed around intestinal calculi after each meal of bran. A calculus of 14 pounds with a diameter of six inches on section showed 720 concentric layers; it would, therefore, have required 360 days for its formation. In a case of Pastore a calculus of the size of a fist had been formed in less than a year.

Aside from the true calculi we find in the large bowel pseudo-calculi (phytoconcrements) and other concrements which lack a definite structure and which owe their formation to pasturing on sandy, marshy meadows, or to marshy feed; also to the habit of some horses to swallow and to nibble on wooden partitions, or to drinking water from shallow brooks or pools, or from wells containing much sand. The latter may form, in the large intestines, mortar-like conglomerated masses with the feces. In the formation of the latter (particularly if much bran is given), phosphates and carbonates of lime, swallowed hairs, dry vegetable parts or swallowed foreign bodies, take a considerable part. (Deysine has reported a case in which he found a swallowed sponge incrustated with lime salts.) The surface of pseudo-calculi and concrements is usually uneven; they often have sharp corners or may be covered by a crust of phosphate of ammonia and magnesia. They are much lighter than genuine enteroliths.

In a case of Grimme several hair balls were found in the large colon of a horse which had been fed for nine weeks with oat meal; they were up to the size of a fist



and had been formed from the hairs of grains of oats. Wiesner found in a three months' old foal a concrement, obstructing the terminal portion of the ileum, composed of food particles, hairs and infiltrating lime salts.

Fecal balls composed exclusively of feces are more rare than concretions. The intestinal lumen of newborn foals may be obstructed by meconium (Töpper).

The lumen of the small intestines may be obturated by animal parasites such as ascaris, gastrus, larvæ (Kater, Rexilius).

The anterior portion of the small intestine is only rarely obturated by swallowed foreign bodies (Bech, Jacobin and Clare, Angebauer, authors' observation). Closure by a hematoma formed in the intestinal wall is also quite rare (Uhlig, Kitt, Schleg & Johnne).

Obturation of the intestines of **dogs** is often produced by foreign bodies swallowed in play or in retrieving (see page 312); also by pieces of bone and cartilage, occasionally also by hair balls, lumps of feces, parasites (tænia); such foreign bodies are much less frequently the cause of obturation in cats.

In **ruminants** and **hogs** obturation of the intestinal lumen by hair or fecal balls, or by swallowed foreign bodies, or by a hematoma (Eber) is rare. (Fetting found in a young heifer, which had died of digestive disturbances, a young cat wedged in the small intestine; Wyssmann saw a case with obturation of the large intestine by masses of fibrin; Spörer saw a case where a potato had become wedged in the small intestine and one case where a vegetable stem formed the impediment.)

**Pathogenesis.** Foreign bodies formed in the intestines (calculi), or arrived there from without, may sometimes remain in a wider portion of the intestinal tract without doing any harm at all. Zschokke found, in a miller's horse which had always been well, forty-two pounds of intestinal calculi, one alone weighing twenty pounds. At other times calculi are the cause of chronic intestinal catarrh (see page 340), or of intestinal stenosis (see page 387). Closure of a narrow portion of the intestines is not at all rare if the bodies have been moved to such places by peristalsis and have there become wedged in; the closure may then be due to the foreign body alone or to it and the masses of feces which accumulate at this site.

The site of the obturation varies according to the derivation of the obturating body. In horses genuine calculi and concretions, also lumps of feces generally become wedged into the first portion of the small colon, more rarely somewhat more towards the anus, occasionally also into the pelvic flexure of the large colon; desiccated feces (chaff) are also sometimes found in the last portion of the ileum or of the duodenum which they obturate rather suddenly (see page 366). The large intestine is usually obturated by fecal agglomerations in dogs. In all other cases (foreign bodies, intestinal parasites) obturation usually occurs in the horse and in all other animals, in the small intestines.

If the obturating body does not stretch the part closed up, because the comparatively large mass is simply held back by the narrowing portion of the bowel without interfering with it, as is, for instance, the case with large calculi situated in the stomach-like dilatation of the colon, then the obturation of the intestinal lumen leads to the same consequences as they are found in fecal impaction (see page 366). If, however, the foreign body has been pushed into a narrower portion of the intestine, it will stretch and irritate the intestinal wall and cause attacks of powerful convulsive contractions, that is, colicky pains. In such and also in the previously mentioned cases, as already explained when discussing the pathogenesis of fecal impaction, convulsive painful contractions of intestinal portions situated nearer to the stomach will occur likewise. If the body wedged in the intestinal lumen is pointed or sharp-edged, it may produce continuous pain.

As in all forms of obturation of the intestinal lumen, the peristalsis becomes abolished behind the obturated point, and often increased for some time in front of the obstruction; later, however, the peristalsis also ceases in the portion between the obstruction and the stomach as stretching of the bowel occurs from the accumulating contents. Bloating occurs only after some time in these portions, if their contents are more or less fluid; the meteorism does not, however, reach a very high degree, because normal intestinal contents do not form much gas and the latter is easily absorbed. In closure of the posterior portions of the large bowel feces collect in front of the obturated point if the appetite has in the mean time remained fairly good; the feces desiccate and extend the lumen of the bowels. Secondary gastric dilatation does not infrequently occur under such circumstances in horses.

Pressure or direct injury exerted by the obturating body not infrequently brings on necrosis or inflammation, and these may extend to the internal layers of the wall, even to the peritoneal covering. Necrosis or convulsive contractions of the intestinal wall may not rarely cause intestinal rupture. As long as complications (inflammation, rupture, possibly high degrees of bloating, dilatation of the stomach) are absent, there are no general symptoms, such as are generally seen after absorption of bacteria and their toxins, after peritonitis and in consequence of severe bloating.

**Symptoms.** In horses the visible signs and symptoms of internal obturation are identical with those of impaction in a part of the cases, particularly if the first portion of the small colon has been closed up (see page 367), except as to defecation and as to the onset of the affection. In obturation the clinical picture develops suddenly in distinction from impaction of feces, and after one or two defecations, complete constipation comes on. In other cases there are severe attacks of colic without any

premonitory symptoms which recur from time to time, usually at shorter intervals. The animals throw themselves down recklessly and roll energetically; exceptionally, when the obstructing concretions have sharp borders, the animals do not throw themselves or roll, because this only increases the pain due to injury by the calculi. The animals betray pain by restless tripping, by looking around towards the abdomen, shaking the head, and occasionally by painful neighing (authors' observation). Abnormal positions, such as dog fashion, kneeling postures, peculiar stretching, may also be frequently observed. They are not of any particular significance, because they are caused by the abdominal tension or by the pressure upon the diaphragm and are frequently seen in the course of other affections.

Examination of the abdomen in obturation of the small intestines shows a decrease or an entire absence of intestinal sounds from the start, and in other cases after a number of hours, eventually only on the third day of the disease.

Directly after the onset of the first symptoms, defecation occurs only a few more times, often only once or twice, then no more feces or intestinal gases are voided, in spite of the fact that the animals make efforts at defecation. If the obturation takes place gradually, diminished defecation occurs for some time, and if the concretions which form the impediment are quite irregular the discharge of thin-fluid feces may be kept up, because these can pass between the irregular surface of the calculus and the intestinal wall, through the clefts which have remained open (authors' observation).

Rectal examination shows that the posterior portion of the rectum is entirely open. If the arm is introduced up to the shoulder, the examiner may feel the obstructing calculus or the concretion, in horses which are not too large, in the first portion of the small colon, immediately in front of the anterior pole of the left kidney; possibly also in the median line. Obturation in the pelvic flexure or in the small colon (so-called abdominal portion of the rectum) can be felt with ease in all horses. An obturation of the ileum or of the dilatation of the large colon can likewise be felt, unless the patients (horses) are very large (see page 367). One can rarely expect to find masses of ascarides in the small intestines.

Calculi and concretions may usually be felt on palpation as hard, unyielding, frequently uneven, nodular bodies, but their true shape may be covered up by fecal masses which have become deposited on them. The intestinal wall at the site of the obstruction is usually moderately tender, but it may be extremely tender, as the authors have seen in one case, so that the patient rears and throws himself down whenever the hand approaches this place. The obturating body may exceptionally be in the rectum, so that it can be felt directly by the introduced hand.

The temperature is at first normal, and respiration and



pulse deviate only very inconsiderably from the normal figures in consequence of restlessness; the pulse remains usually below 50 in the beginning. Obturation of the anterior portions of the small intestine forms an exception from the general rule because the early occurrence of dilatation of the stomach or of enteritis frequently raises the pulse rate shortly after the onset of colicky symptoms. Dilatation of the stomach may cause belching, retching or vomiting.

Obturation due to retention of the meconium on the second day after birth, causes marked restlessness, pawing with the front legs, wagging of the tail, stretching, pressing, lying on the back, dog-fashion squatting, sometimes even convulsions (Töpfer).

If there is no improvement one usually sees in the further course, rarely on the first, more generally on subsequent days, an elevation of temperature; also acceleration and weakness of the pulse. These symptoms increase very slowly; they are caused by the onset of complications (enteritis, peritonitis, meteorism, rupture). Rupture of the intestines is not followed by collapse, provided it has been small and, provided only solid feces have gotten into the abdominal cavity, because then bacteria and their toxins are not at once absorbed in larger amounts; but there develops the clinical picture of general acute peritonitis, characterized by unevenness and tenderness of the peritoneum, which can be ascertained on rectal examination; one may also frequently be able to feel free particles of feces in the abdominal cavity. If an exploratory puncture is made, one obtains a fluid exudate containing particles of feces and numerous bacteria.

The clinical picture in **cattle** is identical with that of intestinal stricture (see page 389) (Holterbach), or one observes more or less marked symptoms of colic, obstinate constipation in spite of repeated efforts. The appetite is poor, rumination has ceased and moderate bloating usually occurs. Intestinal sounds are absent. Rectal exploration sometimes reveals the presence of an obturating foreign body in the intestines, which may be felt in the right half of the abdominal cavity (Spörer, Feser).

In **dogs** we observe obstinate vomiting, possibly colic, absence of appetite, but increased thirst and complete constipation. The animals are less lively, hide themselves, whine and cry on getting up, on moving, and also while at rest; they often change their place of rest, are cross and excitable. In the further course of the disease they become increasingly apathic; there may be convulsions, elevation of temperature, acceleration of pulse, rapid emaciation. Palpation of the abdomen reveals marked tenderness in places, and possibly the presence of the obturating body (the latter may often be easily detected during narcosis). A pointed foreign body (tooth pick) gave rise, after perforation of the intestines, to the formation of an abscess.

**Course.** The disease is usually of short duration in larger animals and in complete obturation; it then ordinarily extends over two to three days, rarely over a longer period; it may, however, occasionally last longer, one to two weeks (authors' observation) or even more. (In a case reported by Felder, the horse died after a sickness of 30 days). In dogs, on the contrary, intestinal obturation often lasts from one to three weeks. Complete obturation is often preceded by the symptoms of intestinal stenosis with occasional, more or less severe, disturbances of defecation or colic.

Recovery occurs rarely spontaneously or only upon internal treatment. It may, however, occur if the obturating body is not too large and can therefore be pressed towards the anus and finally expelled by convulsive contractions of the intestinal wall. Very exceptionally a calculus may return from the beginning of the small colon into the stomach-like dilatation.

In the majority of cases complications lead to a fatal issue, the end being ushered in by febrile temperature, frequent and small pulse, collapse; the intense abdominal pains frequently cease suddenly (rupture) or gradually before death occurs.

**Diagnosis.** Obturation of the intestines can be diagnosed positively only after the obstructing body has been felt, either by rectal exploration or, in smaller animals, through the abdominal wall. By means of X-rays one may often detect the foreign body in dogs. The history may furnish valuable data in dogs, and in horses a history of continued feeding with bran and crushed grain or of a long stay on sandy, marshy pastures, creates the suspicion of obturation by calculi or concretions.

The disease is distinguished from primary fecal impaction by the fact that a part of the cases (of obturation) is complicated by severe abdominal pains; that a distension of the rectum by fecal masses is absent in the beginning and eventually even later, and that there is obstinate constipation from the start. Fecal accumulation in the rectum is not seen in intestinal displacements or in thrombosis of mesenteric vessels, but severe bloating of some portions of the intestines and a weak accelerated pulse are observed early.

**Treatment.** In the horse the expulsion of small calculi may be brought about by laxatives, such as castor oil (500-600 gm. with olive oil, with ether 50-70 gm., or with mucilaginous substances, some *radix liquiritiæ* as an electuary), also pilocarpine (0.20-0.30 gm.). Eserine or chloride of barium should not be used, because they may bring about intestinal rupture. If the calculus is in the neighborhood of the anus, it can be grasped with the fingers of the introduced hand and it may be removed by a twisting motion. Some intestinal concretions can easily be crushed with the hand and can then be removed piece-meal. One may also cautiously attempt the breaking up of a concretion lodged in the first portion of the small colon. If the cal-

culus is situated more anteriorly one might try to push it into the stomach-like dilatation (Colin); this attempt is, however, rarely successful on account of the accumulation of feces in front of the place of obturation, as the authors' observations in this respect have shown. One may also try the injection of large amounts of water (30-40 liters at one time).

Töpper recommends Masch's meconotarium for the removal of meconium which is impacted in the rectum; this instrument has the shape of a spoon and it must be introduced repeatedly with care. After cleaning out the rectum an injection is given (1 quart of 1% Lysol solution) and internally 1.0 calomel with 50 gm. castor oil.

If these attempts fail or if the size and shape of the obturating body exclude the possibility of success from the start, laxatives should not be given, but a laparo-enterotomy might be tried. However, the latter will be successful only in very exceptional cases, since the union of the stomachlike dilatation of the large colon into the small colon is usually displaced into the latter, and it therefore becomes impossible to draw this portion of the intestine into the wound of the abdominal wall, because the former is too firmly connected with the upper abdominal wall. The operative procedure appears indicated only in obturation of the small colon with its long mesentery or in obturation of the pelvic flexure. The left side should be selected for the abdominal incision. The right would be preferable in obturation of the head of the cecum, but this condition cannot be diagnosed intra vitam. Hoffmann recommends the removal of the calculi wedged in the posterior portion of the rectum, by introducing the hand through the laparotomy wound and pushing carefully the stone towards the rectum where it will be grasped and extracted by the hand of an assistant (water should be introduced beforehand if the stone is of any considerable size). Large calculi can, of course, not be removed by this route.

If a large calculus is situated in the stomach-like dilatation so that it occludes the opening of the small colon, it is impossible to pull this part into the laparotomy wound as it is provided with a short mesentery only. Provided there is not yet any considerable fecal impaction one may, however, try to draw the right upper portion of the colon into the wound made immediately below the right costal arch, to incise this portion of the bowel, introduce the hand into it and push it forward until the stone can be grasped and extracted. An attempt of this kind was, however, futile in a case of Plósz & Marek because the upper right portion of the colon was so much filled with firm fecal masses that it could not be drawn into the laparotomy wound.

One can, anyhow, expect success from laparo-enterotomy only if dry feces have accumulated in larger amount in front of the obturating body and in the absence of grave complications, such as enteritis, peritonitis, intestinal rupture. A case of Felizet in which a calculus, the size of a child's head, was successfully removed by an operation made at the proper time proves that it may be carried out to bring relief and recovery. In the cases reported by Richard, Dollar & Rogers, by Hall, Roeder and Lowe, and in three cases of Marek & Plósz the patients operated upon all died.

Obturator foreign bodies in **cattle** may also be removed by operative procedure, as is shown by the observations of Spörer and Feser.



In a case reported by Spörer an obturating potato was crushed after laparotomy had been made, and in another case a rough stalk was broken in the middle; both cases then recovered. Feser successfully removed a piece of tin by laparotomy.

The expulsion of foreign bodies wedged in the intestinal tract is favored in dogs by the administration of laxatives. Foreign bodies in the rectum can be removed by hand or with proper extracting forceps. If these methods are not successful, or if they are not applicable from the start on account of the nature of the obstructing body, laparo-enterotomy may be successfully performed, as is shown by the reports of Felizet, Siedamgrotzky, Fröhner, Degive, Plósz, Marey and others. It is, of course, not advisable to wait long with an operation if it is at all indicated.

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## 22. Narrowing of the Intestine. Stenosis intestini.

Stenosis of the intestines is a more or less chronic disease with a generally progressive diminution of the lumen of the intestine at a circumscribed place.

**Occurrence.** Stenosis of the intestine is a rare affection. It occurs in horses with about the same frequency as obturation of the intestines (see page 379).

**Etiology.** The following are the pathologic conditions which may be the causes of intestinal stenosis: Scar formation in the intestinal wall caused by injury through foreign bodies, by ulceration, possibly after gangrene of an intussuscepted portion of the bowel, produces narrowing of the intestinal wall (strictura intestini) in consequence of cicatricial contraction of the newly formed connective tissue. Upon this basis intestinal stricture is usually found in the small intestine, the small colon, or the rectum, in domestic animals with the exception of the hog. According to Bruckmüller, Sikorski, Bolton, in horses occasionally, but according to the reports of the Berlin pathological institute frequently, a cicatricial stenosis of the ileocecal opening is noted, due to chronic inflammation of the mucosa and caused by the continuous irritation of coarse feed

(short-cut chaff, buckwheat straw). Ligation of the prolapsed rectum often causes stenosis in hogs (Johne).

**Chronic peritonitis** which produces newly formed and subsequently shrinking cicatricial connective tissue on the serosa of portions of the intestines, may cause stenosis of the intestines of variable extent. (Ross, Zürn, Dignac, v. Ow., authors' observation.) Intestinal stenosis is frequently produced in chronic adhesive peritonitis, in consequence of kinking in loops of intestines which have become adherent to the abdominal wall or to neighboring organs, because the two branches of the adherent loop pull upon the place of attachment by their own weight. (Avérous, authors' observation.) Sometimes localized subacute peritonitis may produce intestinal stenosis in this manner, as has been seen in the horse (authors' observation).

**Neoplasms** in the intestinal wall are not infrequently the cause of intestinal stenosis (Achilles found intestinal tumors, mostly sarcomata in 0.2% of the horses slaughtered in the stock yards at Leipzig). Most frequent are polyps of the mucosa; they may be multiple in this place. Sarcomata are usually found in the small intestine; they are either firm or soft, and they also assume an infiltrating form (Rabe, Kitt) with the histologic structure of a lymphosarcoma. Melanomata occur in the cecum and rectum of the horse (Harrison, Csokor). Carcinomata are frequently seen in the region of the anus of the dog (Fröhlner); they are, however, rare in other parts of the intestinal tract and in other animals. (According to the compilation of Achilles only six cases have so far been reported in the horse); these carcinomata in horses have no tendency to undergo ulcerative changes. As rare neoplasms must be mentioned: fibromata, myxomata, lipomata, leiomyomata, adenomata, adeno-carcinomata, actinomycomata (the latter are of course, not true tumors).

Guénon removed from the rectum of a horse a pediculated, very hard tumor the size of a walnut; it was, according to Petit, formed in such a manner that a calculus had formed in a gland of Lieberkühn. This calculus had gradually distended the mucosa and had drawn it out with the formation of a pedicle. In a case of Cadéac congenital valve formation in the rectum had been the cause of stenosis.

**Calculi, balls of feces, foreign bodies**, not infrequently cause stenosis before entirely obstructing the lumen of the intestines. Circumscribed dilatations of the intestinal wall (diverticulum intestini), which may either be congenital (diverticulum of Meckel in the ileum) or acquired through the action of heavy bodies (sand, calculi, fecal balls), and may constrict the lumen of the part where they are located by compressing it when filled or by twisting it through traction. These forms of diverticula are most commonly found in the horse in the last portion of the ileum, in the large intestine, or in the small colon.

**Compression of the intestine** by an abscess or a neoplasm formed in the mesentery or by enlarged abdominal organs.

(gravid uterus, filled rumen, enlarged ovary, enlarged prostata); a spleen enlarged in consequence of hemorrhage may not infrequently cause stenosis of the bowels. Hendrickx saw in a horse a compression of the intestines due to a detached, partly ossified cryptorchitic testicle, which subsequently broke through the atrophic intestinal wall into its lumen.

Intestinal stenosis may also be caused by **internal herniae** before they become strangulated; the clinical picture then usually terminates by the symptoms of complete incarceration. **Parasites** (ascaris, gastrophilus larvæ [Kater, Rexilius], intertwined tæniæ) will only exceptionally produce a picture similar to intestinal stenosis.

**Pathogenesis.** According to the character of the enumerated causative factors, the intestinal lumen usually becomes narrowed very gradually; in exceptional cases the stenosis remains stationary or it only occurs temporarily (diverticulum, internal hernia, compression caused by an overfilled rumen). The narrower the affected portion of the intestine, even under normal conditions, the firmer the feces, the more abundant and the drier the ingested feed has been, the earlier, the oftener, and the longer will the passage of feces through the constricted portion be interfered with. The fecal masses accumulating in front of the narrowed point stimulate the intestines to forcible contractions at the place where the accumulation has taken place; some of these are convulsive and cause colicky pains. These forcible contractions will finally succeed in pressing the accumulated feces through the stenosed portion and the time which elapses before this occurs depends upon the degree of stenosis and upon the consistency of the feces. After the feces have passed the narrowed point, the convulsive contractions and the pains cease. Hypertrophy of the forcibly contracting muscularis and dilatation of the intestine will develop in the course of time. The nearer to the stomach the constriction is situated, the sooner after the ingestion of food will abdominal pains come on, and a chronic dilatation of the stomach will usually develop; such a condition may, however, also develop in stenosis of the large intestine. In the latter condition attacks of pain occur independently of the ingestion of food; in stenosis of the rectum they commonly come on before defecation. Those portions of the intestines which are situated between the stricture and the anus do not contain a great deal of feces, hence they act sluggishly. The feces accumulate and decompose in front of the stricture and frequently become instrumental in producing chronic intestinal catarrh, which influences the nutrition of the animals unfavorably. The stenosis may finally lead to complete occlusion of the bowels.

**Symptoms.** In the horse and in cattle a clinical picture is observed at variable intervals, which is identical with that met with in impaction (see pages 367 and 376); the attacks may last



for a few hours only, or for several days (so-called habitual, recurring or periodic colic). With remitting mild or on the contrary, very severe attacks of colic, there develops afterwards an incomplete constipation. In stricture of the small intestine the attacks usually occur shortly after the ingestion of food, and they are often followed by dilatation of the stomach (see page 299). In stenosis of the rectum restlessness is noticed before defecation. In cattle there occur, aside from the symptoms of intestinal obstipation, those of a periodically recurring atony of the stomach or overfilling of the rumen (Storch).

During the attacks of colic, pulse, respiration and temperature remain, as a rule, normal or are only changed insignificantly; an exception to this rule is noted, however, when a dilatation of the stomach or enteritis has set in, or where the underlying cause of the stenosis of itself leads to fever (abscess, peritonitis).

During the intervals between the attacks the animals either appear perfectly healthy, as for instance in moderate stenosis of the small intestine, or defecation is scanty, occurring only after longer intervals, or instead of being dry and small, the feces may be softer than normal, a condition which is due to secondary intestinal catarrh.

Rectal examination often does not merely reveal the seat of the stenosis but also its cause. In stenosis of the rectum the hand meets an impediment, either near the anus or somewhat more distant from it, which cannot be removed either by spreading the finger or by the injection of lukewarm water (this distinguishes true stenosis from temporary constriction, which can be overcome by the means indicated); one often also can feel feces with the finger pushed through the constriction, sometimes a dilatation and occasionally a kink. If the arm is introduced up to the shoulder, narrowing of the small colon may be detected, because all loops of this part of the bowel are in a portion of the abdomen which can be reached from the rectum. A stricture in the posterior third or possibly the posterior half of the left loop of the large colon, or in the upper half of the cecum, the terminal portion of the ileum, or in the loops of small intestines situated below the renal region, may be detected in the same manner, and in horses which are not too large they may even be found in the stomach-like dilatation of the colon. One finds the intestinal wall thinner at the constricted portion, possibly adherent to a neighboring organ, or one may detect a large abnormal formation in its lumen or in the neighborhood. In front of the stenosis there is usually an accumulation of feces. Palpation of the right half of the abdomen in cattle may show stenosis of the rectum, stenosis or adhesions of the cecum, stenosis of other portions of the large intestines or of some loops of small intestine.

The presence of a non-incarcerated hernia of the diaphragm may be assumed with some degree of certainty from the pres-

ence of marked loud intestinal sounds in the thorax, of tympanitic sounds, varying at short intervals in intensity and pitch, and heard at the posterior portions of the thorax; also from the occurrence of intense dyspnea in walking down steep roads or from lying on the side. Diaphragmatic hernia may also lead to asthmatic symptoms (Bärner). Loud intestinal sounds are also heard over the thorax whenever peristalsis is much intensified and a tympanitic sound over the posterior lower parts of the chest can be heard not infrequently if gas containing portions of the large colon have for some reason or other been pushed towards the thorax and have pressed the diaphragm into it.

Intestinal stenosis in **hogs** and **carnivora** is accompanied by gradually increasing obstipation. Rectal exploration reveals a constriction of the rectum somewhere in the pelvis (tumors, enlarged prostate, enlarged uterus, cicatrix). Palpation of the abdomen shows a firm, hard body in the intestines, or in the mesentery, diffuse adhesions with the formation of lumps or the enlargement of certain abdominal organs.

**Course.** Stricture of the intestines does not lead to any disturbances for some time; after a variable interval of time, however, there occur, in larger animals, particularly in horses, attacks of abdominal pains of variable duration. They recur at first after long intervals, sometimes only after one to two years, but they become more frequent as the disease progresses. The intervals depend to a certain extent upon the nature of the feed and upon the amount of work required of the animals (Bärner). As the attacks become more and more frequent and as obstipation increases continually, complete and permanent intestinal obturation may occur. In the mean time the animal has become emaciated and death occurs from enteritis, peritonitis, rupture or displacement of the intestines, exhaustion. In some cases an animal in a fair state of nutrition may succumb, during one of the first attacks, to rupture of the stomach or intestines, or to enteritis.

**Diagnosis.** Stenosis of the intestines and its origin can only be positively diagnosticated by rectal exploration or by palpation of the abdomen. In the absence of positive findings the presence of stricture of the intestine may be assumed with a certain degree of probability from the history of certain preliminary diseases, leading to narrowing of the lumen of the bowel, and leading in shorter and shorter intervals to attacks of colic with constipation, especially after the ingestion of dry or firm feed; at the same time general symptoms are absent, and a gradually increasing obstinate obstipation develops in smaller animals. However, the symptoms here enumerated do not permit the exclusion of fecal impaction or thrombosis of intestinal vessels from other causes.

**Treatment.** In the majority of cases the treatment recommended against fecal impaction is indicated (see there). In the cases of narrowing of the intestine, which are due to a non-incarcerated hernia or to an excessively large foreign body, it is not advisable to use laxatives with intense action, because incarceration of the hernia or complete occlusion by the foreign body may occur. In such cases one should confine oneself to the use of the neutral salts. In rectal stenosis, local treatment may be assisted by mild laxatives. If dilatation of the stomach has occurred the use of the stomach tube and lavage of the stomach are indicated.

If these methods are not successful, or if an improvement cannot be expected from them in consequence of the nature of the stenosis, relief may be attempted by operative procedures.

Pedunculated tumors of the rectal wall should be removed by crushing or ligation of the pedicle; tumors with a broad basis or those of the paraproctal connective tissue must be removed by excision. Periproctal abscesses must be properly treated after an incision. Douville removed a cyst from the rectum of a horse by puncture, followed by injection of Lugol's iodine-iodide of potash solution. Certain forms of stenosis situated in front of the pelvis may be attacked through the rectum (tearing of adhesive bands, displacement of enlarged organs). (Guitard temporarily relieved in this manner, a stenosis caused by an enlarged rumen.)

The great majority of cases of stenosis formed in parts that are distant from the pelvis can be treated only by a laparotomy or eventually an enterotomy. It is absolutely necessary first, to ascertain the exact nature and seat of the stricture by rectal exploration, because laparotomy is a dangerous operation, particularly in the horse, and its successful performance requires the selection of the proper place for the incision; the latter should, if possible, be made at a place nearest to the site of the intestinal stricture. In smaller animals, however, the best place for the laparotomy incision is, as a rule, the median line (linea alba). Stenosis caused by adhesive bands can be removed most easily, because all that is generally necessary after laparotomy is the severing of the constricting bands. Plósz & Marek have successfully performed laparotomy in such cases. Deghilage broke up adhesions of the small colon with the hand introduced through an incision in the vagina. De Meis & Parascandolo have removed a carcinoma of the cecum in a dog by laparo-enterotomy.

Proper regulation of diet (green feed, bran slop, thin flour pastes, milk, occasionally molasses) usually postpones the development of complications and of an unfavorable termination.

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### 23. Obstruction of the Mesenteric Arteries. Thrombosis et embolia arteriarum mesenterialium.

(*Verstopfung der Gekrösarterien; Thrombotisch-embolische Kolik* [German]; *Congestion intestinale* [French].)

Thrombosis of the mesenteric vessels is due, as a rule, to inflammation and obstruction, caused by *sclerostoma larvæ*; it occurs in this connection only in horses, and leads often to frequent temporary attacks of colic.

Many French authors classify intestinal disturbances due to thrombosis and embolism with "*Congestion intestinale*," to which also belong catarrhal and inflammatory conditions (see page 337). This cannot be approved, since it throws very heterologous affections together under one head.

**Historical.** Larvæ of *sclerostomum* were first seen in the mesenteric arteries by Ruysch in 1665 and similar observations were then made by several other authors. The causal connection between these parasites and attacks of colic has since the beginning of the last century been pointed out by several authors, especially by Rigot, Schutt, Hering, Reynal, Bruckmüller, Prehr and others. Bollinger (1870) made systematic examinations of this pathologic condition. Modern investigations of verminous aneurysm of the horse have more recently been made by Sticker (1901), Glage (1905), Adelman (1908). Disturbances of the circulation of the blood and of the functions of the intestines following thrombosis of the mesenteric arteries have been studied by Marek (1907).

**Occurrence.** Bollinger's investigations, which have been confirmed by other authors, have shown that 90 to 94% of all horses with the exception of foals have a verminous aneurysm. However, the statement of this author must be considered as entirely unjustified, that one-half of all fatal cases of colic are caused by thrombosis or aneurysm and that all cases in which colic appears suddenly and without any apparent cause are due to this condition. More recent observations (Matthiesen, Franke, Schultze, Pöppel, Glage) prove that the disease also occurs in foals from the third month on. Pöppel, indeed, has seen, in a foal ten days old, a dilatation of the anterior mesenteric artery as large as a pigeon's egg, and in it a thrombus containing larvæ of *sclerostomum*.

Verminous aneurysm appears to be more common in foals than is usually assumed. Among thirty-five slaughtered foals, from five to several months old,

Glage found aneurysm of the anterior mesentery in fifteen, varying in size from a walnut to a fist.

The following figures about the frequency of thrombotic infarction of the intestinal wall are given from various sources: Dresden Clinic, 1899-1908, in horses dead after attacks of colic, 9.5%; Berlin Clinic, 1891-1907, in 3%; Prussian army, 1892-1908, 6.6%; Budapest Clinic, 1900-1909, 14.4%. Wall claims that thrombotic-embolic disturbances of the intestines are quite rare. Indeed there seem to be great differences in the frequency of the affection, according to periods of time and to various countries.

**Etiology.** According to the investigations of Sticker, thrombosis of the mesenteric arteries of the horse is caused by the larvae of *sclerostomum vulgare* Looss (*Scl. bidentatum* Sticker).

The mode of the migration of the *sclerotomae* larvae into the intestines and from there into the mesenteric arteries of the horse is at present not yet known with certainty. According to Leuckart and Willach, the ova of the mature worm, inhabiting the large intestines of the horse, are voided with the feces, and from these free ova the larvae are liberated in water, on moist walls of barns, in manure. From here they get again into the horse with drinking water, or with food contaminated by manure, etc., and on moist marshy pastures. The correctness of this view is confirmed by the observation of Mieckley, who prevented the appearance of verminous aneurysms in the foals of the Beberbeck stud by having them drink only filtered water; Ostertag found in the unfiltered water of this stud *strongylus* larvae. Other authors claim that the embryos are already set free in the animal host.

It is claimed that when later on the larvae bore into the intestinal wall, some of them may quite accidentally get into the veins and with the blood current into the liver, from where they reach the lungs, the larger circulation, or the mesenteric arteries. (Colin, Willach, Olt.) According to other authors, however, the larvae get into the arteries in their regular cycle of development (Leuckhart, Railliet, Sticker, Glage). Pöppel believes that intrauterine infection of the embryo is possible, because he once found a verminous aneurysm of the size of a pigeon's egg in a ten-days-old sucking foal. He thinks that this mode of infection may easily occur, since the embryos of *sclerostoma* are microscopic in size (see also chapter on palisade worms).

In horses as well as in other animals there occurs exceptionally a transportation of emboli into the mesenteric arteries in consequence of cardiac disease, endocarditis and aneurysm of the thoracic aorta. The emboli in the mesenteric arteries are usually not followed by serious consequences on account of their small size. The authors have seen the case of a dog where an ulcerative endocarditis was followed by an embolic closure of arteries of the small intestines, and this led to hemorrhagic infarct of the intestinal wall. A. Koch and Leibenger have each seen a case of aneurysm of the anterior and posterior mesenteric artery in the hog.

**Pathogenesis.** The larvae of *sclerostoma*, according to general belief, adhere to the intima and even bore into it, after they have gotten into the mesenteric artery; this causes inflammation

and the formation of blood coagula, also after some time, a thickening and a dilatation of the vessel wall. Sticker holds a different view, namely, that the larvæ become lodged in the vasa vasorum, cause infarction in the media of the vessel, and then mesoarteritis, or peri- and endarteritis. These changes are seen most frequently in the anterior mesenteric artery, or in the art. ileo-cæcocolica, rarely simultaneously or exclusively in a branch of this artery; also in the posterior mesenteric artery or in the art. cœliaca.

In 108 cases of aneurysms in horses Hering found the art. mesent. involved 100 times (92.6%), the arteria cœliaca five times (4.6%), the art. mesent. post. twice (1.8%) and the art. renalis once (0.9%). Bollinger's sixty cases were distributed as follows: Fifty-three times (83.3%), the art. mesent. thirty-two times in the main trunk; ten times each in the upper and in the lower branch; once in a branch of the small intestine; twice (3.3%) in the abdominal aorta; twice (3.3%) in the art. cœliaca; once (1.6%) in the art. mesent. post.; and twice (3.3%) in the art. renalis. In twenty-nine cases of aneurysma in the art. mesent. ant., Wall saw the aneurysm in the main trunk of this artery only once (3.4%); in the other twenty-eight cases (96.6%) it was found in the art. ileo-cæcocolica. Adelman found in all of his ninety-five cases the aneurysm in the art. mesent. ant.; in seventy-three horses this artery and its branches alone were involved; in one horse the art. mesent. post. was affected simultaneously; in four horses the posterior aorta; in two horses the art. cœliaca; in one horse the portal vein; in one horse the splenic artery and in three horses the pulmonary arteries. In these eighty-five cases thrombosis was usually seen in the art. ileo-cæcocolica and also sixteen times simultaneously in the art. col. ventralis; ten times in the art. col. dors.; seven times in the art. ileo-cæcalis, and three times in an artery of the small intestine.

The formation of a thrombus leads to a narrowing of the lumen, provided that dilatation is not commensurate with the diminution of caliber, or if the end of the thrombus projects into the lumen of a branch. The diminution of the lumen rarely goes on to complete obliteration in a main trunk, but complete obstruction of lateral branches occurs somewhat more frequently.

Stenosis or complete obstruction of mesenteric arteries may be brought about in a variety of manners. It may occur that a thrombus formed somewhere increases gradually in size until it closes the lumen completely. The more rapidly the thrombus grows, the earlier may disturbances of circulation come on after complete obstruction. There is also the possibility that two or more neighboring and anastomosing arteries are affected simultaneously by thrombosis; in such a case even a stenosis of not very high degree may become dangerous. One cannot infrequently see that in non-obturator thrombosis of the art. ileo-cæcocolica or of the art. colica ventr., blood can get in sufficient amount into the colon through the arter. colica dorsalis, so that its blood circulation remains fairly normal. If, however, the art. colica dors. becomes stenosed or completely obstructed, local circulatory disturbances will come on without fail. Cases are not rare in which a thrombus formed in a main trunk will become elongated, and will reach and obstruct the opening of an arterial branch. A thrombus formed in the art. ileo-cæcocolica and growing peripherally may reach or even



grow into and obstruct the art. colica ventr., or one of the art. cecales, or the ramus iliacus; if a thrombus in the art. ileo-cæcocolica grows centrally, it may spread into the art. colica dors., or into one of the arteries of the jejunum. In such cases, according to the observation of the authors, obstruction of the branch occurs in the following manner: The blood current moves the free floating end of the thrombus and eventually presses it into the opening of one of the branches of the main trunk. If the terminal free end has once been pressed into an opening, then closure is intensified, because there is a decreased blood pressure peripherally and an increased blood pressure centrally. However, if a sufficient collateral circulation is established, the blood pressure again may become evenly balanced in front of and behind the obstruction, and the thrombotic plug may again become free.

In the larger mesenteric vessels thrombosis may bring about embolic closure of smaller branches. Since such emboli are, as a rule, small, and since they therefore obstruct only smaller arteries, they do not, as a rule, produce any serious disturbances of circulation, either alone or in connection with the primary thrombus. Only if arranged in arteries one above the other, like the floors of a building, might emboli become dangerous. No doubt every thrombus does not necessarily lead to embolus formation. On the other hand, thrombosis in other organs or in the aorta, may lead to embolism in the mesenteric arteries.

**The effect upon the circulation in the intestines of obstruction of the mesenteric arteries** depends mainly upon the size and the number of the obstructed vessels, upon the seat and the degree of stenosis and upon the size of the anastomoses which connect neighboring vessels with those that are thrombosed.

The *arteria mesenterica anterior sive cranialis* (Fig. 40 b.) supplies all portions of the intestines with blood, except the oral portion of the duodenum, which is supplied by the art. cœliaca, and the middle and caudal third of the small colon (so-called abdominal portion) of the rectum, which get their blood from the art. mesent. posterior, s. caudalis (n). The *arteria mesent. ant.* forms a short, strong trunk, only a few centimeters long; this gives off, in front and somewhat to the left, the art. colica dorsalis (d), which supplies all upper branches of the colon, and the art. col. media (e), which supplies the cranial third of the small colon. The two last named arteries usually arise from a common trunk (c); somewhat more backward and to the left arise the 17-20 jejunales (i), which supply the small intestines. In this manner the main trunk becomes thinner and is now known as the art. ileo-cæcocolica (f); it is two to four cms. long; it first gives off the arter. colica ventralis (g), which supplies the lower branches of the colon; the remaining portion, the art. ileo-cæcalis, divides into the art. cæcalis (h), lateralis (k) and the ramus iliacus (m) to supply the cecum and the ileum. The arter. cæcalis lateralis gives off a branch to the lateral wall of the right lower branch of the colon (art. colica lateralis of Franck).

The most cranial artery of the small intestines (art. jejunalis) anastomoses with the arter. pancreatico-duodenalis, that is, with the art. cœliaca in the region of the duodenum, by a tolerably large arterial arch situated in the mesentery. The different arteries of the small intestines communicate with the ramus iliacus by similar archlike anastomoses (j). The art. colica ventralis and the ramus iliacus are connected with the vessels of the cecum which among themselves possess rich anastomoses. The art. colica dorsalis is continued immediately into the art. colica ventralis at the pelvic flexure, and this forms the largest anastomosis of the animal

body (*h*); the art. colica ventralis also forms anastomoses with the art. col. lateralis. If one adds that there are anastomoses between the art. colica dorsalis and the art. colica media, and also between the latter and the cranial branch of the art. mesent. post. (*e*) and between all branches of the latter (*o*), one can easily see that blood can get abundantly into the territory of the branches of the art. mesent. ant., either from the art. colica or from the art. mesent. post. and into the art. mesent. post., and also from neighboring vessels, even if either one of the arteries named is obliterated and the pressure in it is decreased. It is also important and should be emphasized that intestinal branches, whether they arise from main trunks or from arterial arches, and the branches in the intestinal wall in the mesentery are connected by rich anastomoses, as can be seen in Fig. 40.

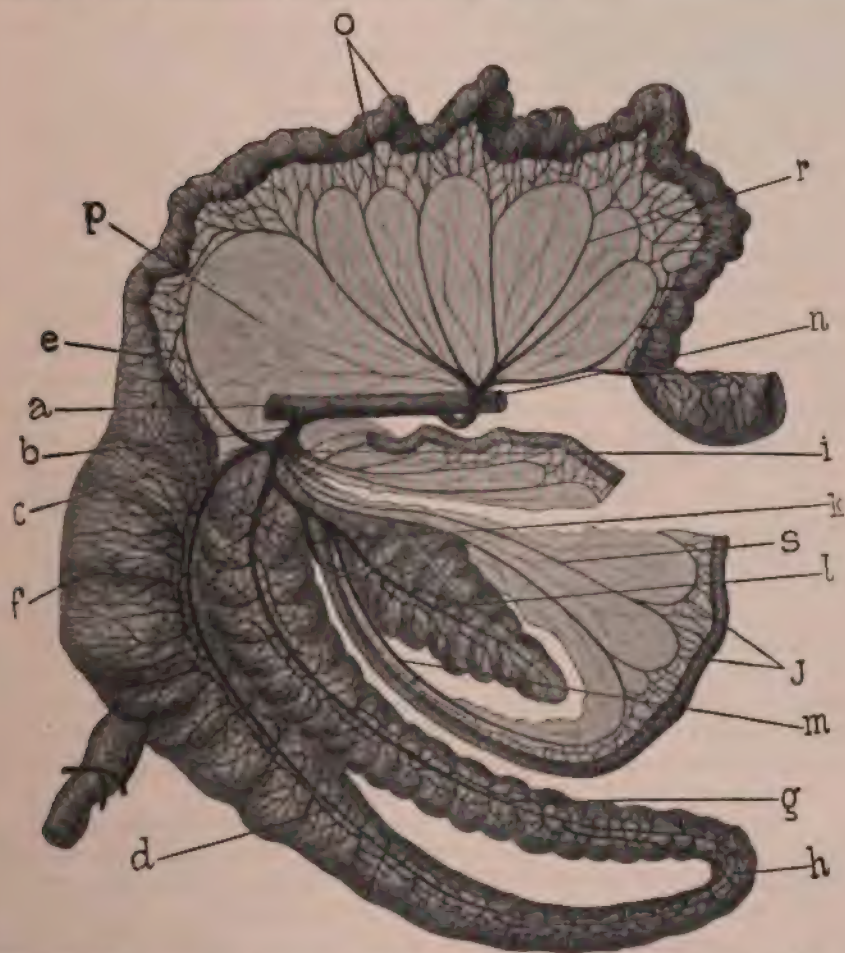


Fig. 40. The mesenteric arteries of the horse. (Adapted partially from Ellenberger and Baum; the vascular anastomoses are drawn after a Teichmann-Preparation.)

Narrowing or closure of the lumen of the mesenteric arteries produces a disturbance in the blood circulation of the intestines only if the arterial pressure in the affected portions of the intestines becomes markedly lower and if consequently the current in the capillaries becomes very much decreased. Marek's animal experiments have shown that total and sudden closure will not be followed by recognizable disturbances of circulation,



filled by blood from the collateral circulation, and this causes more or less marked reddening of the mucosa, which is followed by hemorrhage.

According to the degree of lowering of the blood pressure, two types of circulatory disturbances in the thrombotic parts may be distinguished; there are, of course, no sharp boundary lines between these two types. In the milder form the occlusion is comparatively small, considering the amount of arterial blood which can still get into the affected territory; the decrease in the velocity of circulation is not very high; hence the extravasation of the blood elements is insignificant and a nutritive disturbance, which can be seen macroscopically does not occur. In the mean time, the arteries supplying the affected territory become dilated, the blood supply soon becomes sufficient, pressure and velocity of current become normal again, generally within a few hours, and the extravasated elements of the blood are absorbed. In the milder form, therefore, there is always a restitution of the blood circulation to normal conditions.

The severe form is characterized by very grave nutritive disturbances of all the tissue elements of the intestinal wall, preferably of the walls of the capillaries and of the mucosa. In consequence of the very considerable retardation of the blood current, the permeability of the capillary walls increases from the second quarter of an hour on, and the blood elements extravasate more and more, in increasing amounts, first into the loose tissue of the mucosa, later also into the other tissues of the intestinal wall, to get finally into the intestinal lumen and into the free abdominal cavity. A backward accumulation of venous blood does not take place. A retardation of the lymph current follows the decrease of pressure in the capillaries and the former produces an accumulation of serous fluid in the lymph spaces of the intestinal wall. All this produces an edematous-hemorrhagic infiltration in the intestinal wall (infarctus hæmorrhagicus). With the profound disturbance of circulation there develops a necrosis of intestinal villi, beginning at the free end and progressing towards the deeper layers of the mucosa. Necrosis of the mucosa may become manifest after two hours, and the deeper layers show the earliest necrotic changes after twenty-four hours; this indicates that contact with the intestinal contents plays a part in the production of necrosis.

In some few cases of the severe type the circulatory disturbances may disappear, but this, of course, takes a much longer time than in the milder form, sometimes several days. In obstruction of some arteries of the small intestine, a re-establishment of the normal circulation may occur in only the two terminal portions of the bowel, while the hemorrhagic infarct persists in the median portion. The limit up to which the circulation may be reestablished after a sudden arterial occlusion is as follows: Neighboring arteries of the small intestines



ly into the blood, or not at all, they rapidly bloat the affected bowel (circumscribed, local meteorism), and this occurs easily since the irregular contractions taking place here and there in the intestinal tract are not favorable to a regular passage of the intestinal contents towards the anus.

The conditions for an active development of gases are, however, not given in mild forms of hemorrhagic infarction, hence no cessation of the erstwhile marked peristalsis occurs, if, after a certain interval, which is usually not long, the equilibrium of circulation is reestablished. On the other hand, grave forms of circulatory disturbances produce, in their further course, a gradual diminution of peristalsis which leads to complete cessation within several, occasionally within one to two, hours.

With the onset of circumscribed intestinal paralysis, peristalsis also ceases in the parts situated posteriorly, usually before the feces from the diseased portion have reached the rectum. The paralytic portion has the same effect as a portion obstructed from some cause or other, and as soon as its peristalsis has completely ceased, portions of the intestines anterior to it are stimulated to convulsive contractions by their accumulating contents; these are usually not very intense and not very prolonged. Finally the increasing contents produce gradual cessation of contractility, which progresses towards the stomach.

The effect upon intestinal peristalsis of circulatory disturbances due to thrombosis of the mesenteric arteries has been explained in various ways. Panum, Cohn and Bollinger assumed that the thrombotic territory was paralyzed from the start. However, this claim was not borne out by observations made in man. Bloody stools have been observed not infrequently in man during the whole course of the affection, and similar observations have been made by Litten and Marek on experimental dogs. However, if there was at once a circumscribed intestinal paralysis, such occurrences would be impossible. Indeed, animal experiments have shown beyond doubt that the immediate effect of the circulatory disturbances is not at all an intestinal paralysis, but intermittent convulsive contractions, producing colicky pains. Kader states that after ligation of the mesenteric arteries in dogs and cats peristalsis ceases after four or five hours. In his experiments, where external influences inhibiting intestinal movements were excluded, Marek found that immediately, or one or two minutes after ligation of the artery, the whole length of the affected bowel performed energetic convulsive movements simultaneously in several places. These active contractions continued until the death of the animal occurred (the animals were not kept alive longer than eight hours); and they even continued for an hour and a quarter if the cadaver was kept in a warm box. If the intestinal loops were so placed that some were hanging down and soon became filled with intestinal contents, their movements ceased sooner. If the arteria mesent. ant., some of its branches and all of its anastomoses had been ligated, and hemorrhagic infarction and in some part anemia had been produced, the movements in the anemic portion ceased after one and one-half hours, while they were still going on in the infarcted territory.

The intestinal wall which is damaged in its nutrition, or eventually necrotic through grave circulatory disturbances, early permits intestinal bacteria to enter into the peritoneal cavity; these then multiply rapidly in a serous exudate and produce circumscribed, later general peritonitis, which causes continuous inflammatory pain; the bacteria and their toxins are also absorbed into the blood, general infection or intoxication

with their symptoms follow; some of the symptoms seen in certain cases may also be due to bloating of larger portions of intestines.

What has been said in the preceding pages explains why thrombosis of mesenteric arteries, though common in consequence of the factors which cause it (sclerostoma), comparatively rarely lead to recognizable functional disturbances. The statement of Bollinger that three-fourths of all cases of colic are due to aneurysma of the mesenteric arteries is beyond doubt exaggerated. A thrombotic-embolic affection as the cause of colic is only diagnosticated in about 15% of the cases of colic seen in the Budapest Clinic (see also page 393).

**Anatomical Changes.** Aside from complications like enteritis or intestinal displacements those cases terminate fatally in which the circulatory disturbances are not compensated. In a certain portion of the intestines, generally in the colon, more rarely in the cecum, or in both simultaneously, or in the small intestines and exceptionally in the small colon, we find on post-mortem examination the mucosa and frequently also the serosa dark blackish red, the mucosa forms pendulous projections, the submucous tissue and the neighboring mesentery are more or less infiltrated with a reddish-yellow serum and considerably thickened (1-3 cm). Similar infiltrations are seen on the exterior layers of the bowel-wall. The affected portion is, as a rule, sharply defined, both from the oral and from the caudal healthy portions, and the boundaries usually correspond to the territories supplied by arterial branches situated above the thrombosed point (see page 397). The intestinal contents in the affected portion and in different cases at a variable distance towards the anus appear dark tarry, bloody, sometimes only reddish and occasionally of normal color, but always thinner. The surface of the mucosa is covered by branlike dead shreds of epithelia, sometimes also with masses of fibrin. In grave cases the necrosis may have spread into the serosa and then yellowish spots can be seen from the outside. Exceptionally only do some portions of the intestine show a yellowish or greenish discoloration in consequence of anemic necrosis (in a case of Casper a part of the rectum, and the small colon, were necrotic). Yellowish or reddish-yellow serous fluid, often also a fibrinous exudate is found in the peritoneal cavity. The evidences of rupture of the intestines or stomach may also be present.

Then there are found thrombi or emboli in the trunk of the mesenteric artery or rather in the art. ileo-cæcolica, more rarely in one of the arteries of the colon or in the arteriæ jejunaes, rarely in the posterior mesenteric artery; emboli are usually found in the smaller branches. The blood coagula are sometimes fresh in appearance, rather moist and elastic, at other times drier and firmer, strongly adherent to the intima; sometimes a number of thrombi are found in the same vessels follow-



ing each other. It occurs occasionally that a thrombus is not found on postmortem examination as it existed during life, because a movable occluding portion of the thrombus may have been moved by section of the affected vessel from the opening which it closed during life. The obstructed vessel, particularly often the anterior mesenteric or the arteria ileo-cæcocolica shows chronic arteritis with thrombosis and usually also aneurysmatic dilation. The blood coagulum usually contains the sclerostoma larvæ, sometimes 100 to 300 individuals, especially in younger animals, while thrombi in older animals contain few or even no larvæ at all. Sclerostoma larvæ are usually less numerous in city horses than in horses recently pastured (Wall).

Contrary to the view of Sticker that one finds larvæ in different stages of development according to the season in which the postmortem examination is made, Glage has shown that larvæ and mature worms may be found at all seasons in the intestines or in the mesenteric arteries of the horse.

**Symptoms.** The disease sets in suddenly with symptoms of colic without any discoverable external circumstances; the animals may be at a meal, at work or at rest. The authors' numerous observations, however, seem to indicate that the disease makes its onset more frequently during work than at other times. This may be caused by the fact that the blood circulation is more active during work and hence the detachment of pieces of a thrombus may more easily occur. The symptoms of colic are either intense from the start, manifesting themselves in reckless rolling and throwing, or they are at first mild and reach a higher intensity later on, or the great restlessness first shown later yields to a more quiet behavior. The attacks of pain occur after shorter or longer, sometimes after very long, intervals. The other symptoms vary according to whether compensation is established within a certain time, or whether this is impossible. One may distinguish clinically a mild and a severe type, depending upon this circumstance.

In the **mild type**, aside from the attacks of restlessness occurring at unequal intervals, a variable intensification of the intestinal sounds is observed in certain regions, sometimes over a large portion of the abdomen, and frequent defecation. The feces are normal except for their generally softened consistency.

Rectal exploration may reveal a pulsating dilatation or a trembling of the wall of the anterior mesentery artery or of one of its branches, occasionally also of the posterior mesenteric artery; the condition of the intestines and of the other abdominal organs is normal.

Pulse and respiration do not show any deviation in frequency and quality, the sensorium of the patients is perfectly normal in the intervals between the attacks of pain.

The restlessness becomes diminished, usually after a short time, always during the course of the first day and recovery oc-



curs. In the **severe type** signs of restlessness, differing in intensity and the intervals between them, are likewise observed; they are, however, accompanied by unnatural positions, particularly in consequence of early bloating. The abdomen often becomes distended at the beginning of the affection, because circulatory disturbances develop first in the large intestine; the small intestines and the small colon do not increase the abdominal circumference, even if bloated, or only very slightly on account of their comparatively small size. The intestinal sounds are loud in the beginning for one or two or sometimes for several hours; they become less frequent and disappear entirely; the more rapidly the bloating of the intestines develops and the more profound the circulatory disturbances, the earlier do the intestinal sounds disappear. The percussion sound either remains normal or becomes louder in consequence of the bloating (see page 360).

According to the behavior of the intestinal contractions defecation may be observed for a few or even for several hours, but then constipation becomes complete and feces and gases are no longer discharged. The feces are softer and in very rare cases mixed with blood. The authors saw blood in the feces in only three out of more than 200 cases of thrombotic-embolic obstruction.

Rectal exploration shows a high degree of bloating in some portions of the intestines (*meteorismus localis s. circumscriptus*), usually affecting the colon, the cecum or both simultaneously or some loops of small intestines. The affected bowel is much dilated, its wall very tense, elastic and not painful. In bloating of the colon its left lower portion is enormously distended, not rarely reaches as high as the left kidney and displaces the left upper portion towards the median line or on the contrary towards the left, sometimes even more or less downward, especially when the cecum is bloated simultaneously. The pelvic flexure is displaced into the pelvis or into the right half of the abdominal cavity and the longitudinal bands of the left lower portion of the colon run from left to right, occasionally in a perfect spiral arrangement. The base of the bloated cecum reaches into the left half of the abdominal cavity and posteriorly into the entrance of the pelvis; the base of the cecum can be recognized as such by a longitudinal band running from the right flank at first backward, then downward and to the left, finally towards the thorax. The bloated loops of small intestines present as arm-thick tense, elastic, sausagelike formations, also the small colon; the latter, however, displays a longitudinal band along its surface. The other loops of intestines usually preserve their normal size until the end in contradistinction to the bloated bowels. The findings in the mesenteric vessels are the same as those in the milder form of the affection.

The pulse becomes weaker one to two hours after the onset and rises to sixty beats per minute. Respiration and

pulse become accelerated and forced (in bloating of the large intestine the respiration is interfered with by the displaced diaphragm. The temperature [Fig. 41] is normal at the onset; it rises soon, however, in consequence of the appearance of complications [peritonitis, general sepsis, enteritis].)

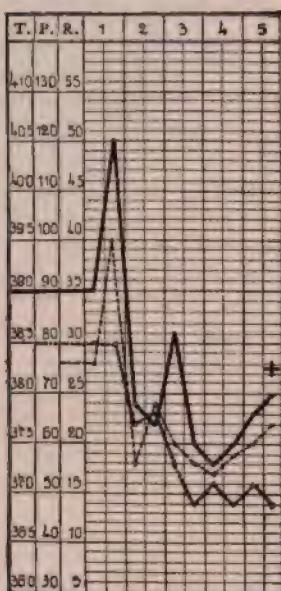


Fig. 41. Fever curve in thrombosis of the mesenteric arteries.

While restlessness persists, becomes less, or even ceases entirely in the further course, the pulse becomes gradually weaker, the respiration becomes more and more forced and difficult, unconsciousness increases, tremor of the muscles and staggering set in, finally the animals fall down and the end comes with convulsions.

In rare cases the severe type of the disease takes a protracted course. If the circulatory disturbances persist for several days without reaching a very high degree, the absorption of intestinal gases will not be very much interfered with, and there is neither an abundant exudate, nor particularly active fermentation of the intestinal contents. Since the nutritive disturbances of the intestinal mucosa do not attain a very high degree, a diffuse peritonitis does not develop, and complete paralysis of the bowel either appears very late or not at all.

In cases of this kind restlessness may be observed, but rarely unnatural positions. The circumference of the abdomen is increased not at all or only very moderately. The intestinal sounds, which are at the onset very marked and of long duration, later on become less frequent without, however, ceasing entirely, therefore the expulsion of feces and gases is not entirely suppressed. The feces are sometimes hemorrhagic and fetid, and now and then intestinal gases are expelled sparingly. Rectal examination shows insignificant or moderate bloating, and in the further course occasionally the accumulation of more abundant masses of firm feces. Pulse, respiration and temperature show some deviation from the normal without, however, reaching anything like what is observed in the severe type and in cases with a rapid course (Fig. 42). It also may happen that the temperature rises considerably at the beginning, and that pulse and respiration become quite markedly accelerated, to decrease considerably later on (Figs. 41 and 42). The sensory functions are clouded, the appetite is suppressed or poor.

The clinical picture lasts sometimes for several days, exceptionally over a week. Friedberger observed in one case a duration of twenty-two days; Kremp one of twenty-one days;

the authors saw in one of their cases a duration of sixteen days before the fatal termination. The protracted form may end in recovery or lead to death in consequence of enteritis, peritonitis or general sepsis.

In rare cases displacement of the intestines is seen in either one of the different types. In severe cases acute dilatation of the stomach or rupture of the intestines may occur.

**Course.** The mild form of the disease lasts from a few quarters of an hour to several hours, rarely longer, never more

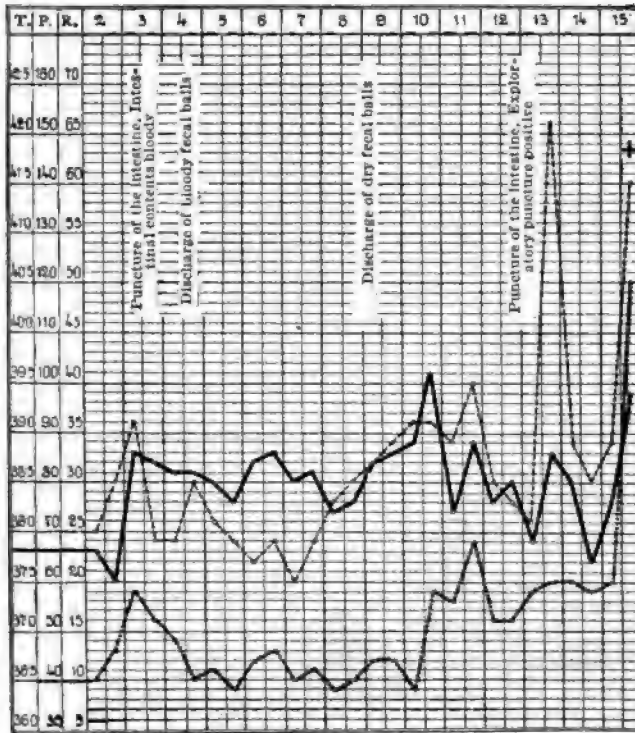


Fig. 42. Fever curve in obstruction of the mesenteric arteries. Fatal outcome. (Protracted course.)

in one day. The severe form has a duration of one to two days, not infrequently only several hours; rarely more than two days and exceptionally two to three weeks, in the latter case it is characterized by attacks of colic which come on after long intervals. Sometimes mild cases of short duration follow each other at such short intervals (so-called habitual, periodic, recurring colic) that one may at first sight gain the impression that he is dealing with a single protracted attack.

The mild form ends in recovery, provided that a displacement of intestines or enteritis have not occurred; but such complications are rare in the mild type; the severe type usually ter-



Milder cases may be confounded with acute intestinal catarrh or with so-called convulsive colic, and the differentiation may not always be absolutely positive. The occurrence of symptoms of restlessness after errors of diet, after exposure to cold, lively intestinal sounds being audible over the whole of the abdomen, fetid soft feces with poorly digested particles of food point to a catarrhal origin of the colicky pains. If such symptoms are not present, however, the differential diagnosis may present great difficulty. The grave forms of mesenteric thrombosis may be mistaken for torsion, volvulus, incarceration, or primary intestinal meteorism. The two former conditions may be excluded by rectal examination, aside from a difference in anamnesis of the case, by frequently occurring, often continuous attacks of restlessness and by complete constipation, which usually supervenes very early. Primary bloating develops after the ingestion of easily fermenting food and is characterized by distension of all of the intestines. (*Meteorismus universalis*.) Without rectal examination, one may confound the affection with intestinal stenosis or fecal impaction. One should, however, in every case of colic consider those disturbances which might be referred to thrombosis of the mesenteric arteries.

**Treatment.** Considering the nature of the disease, the object of treatment can only be the production of collateral blood circulation or the diminution or removal of the effects of the disturbed circulation. The establishment of a collateral circulation should be attempted by walking the animals and by the subcutaneous injection of camphor oil (every two to three hours 20 to 50 gm. up to 250 gm. pro die). Caffeine (5 to 10 gm.). Occasionally the intravenous infusion of physiologic salt solution is indicated. In view of the possibility of detaching portions of thrombi by increasing the velocity and pressure of circulation, and in view of the fact that a collateral circulation is easily established in the milder cases without any assistance from without, this procedure ought to be reserved for the grave cases, in which an additional embolism is a lesser evil than a continuous grave circulatory disturbance. Venesection with bloodletting which is in great favor with French veterinarians can only do harm, since it leads to a decrease of blood pressure.

Subcutaneous injections of morphine (0.4-0.5 gm.) or rectal injections of chloral hydrate (25-50 gm.) appear indicated in all forms of the disease, first in order to relieve abdominal pain, and second in order to suppress violent peristalsis, to prevent the possible occurrence of intestinal displacement or of rupture of the bowel. Breton recommends in colicky pains intraperitoneal injection of chloral hydrate 25 to 30 gm. in ten parts of sterile physiologic salt solution, but this procedure can be dispensed with and indeed it appears quite objectionable.

In the mild form one should abstain from the administra-

tion of any other drugs except narcotics. In the grave form puncture of the bowels should be made as early as possible to prevent the occurrence of complete paralysis and to remove gases from the intestines, which are irritating the latter and positively endanger the life of the animal if they fill larger sections of the bowel.

Laxatives are useless and may be positively harmful. Stimulation of peristalsis is not necessary in the milder forms, while no procedure possible will stimulate peristalsis in the affected bowel in the graver type of the disease. Laxatives may, as already said, be harmful, because they will produce increased movements everywhere in the milder form, and in the severe type in those parts of the intestines which are situated in front of the affected portion. These movements increase the abdominal pain and convey masses of feces into the paralyzed portion; they increase bloating and accelerate the development of inflammation or rupture. Mild laxatives may possibly be given in the graver cases during the period of recovery.

Resection of a portion of intestine and suturing of the severed healthy ends may be tried in disease of shorter portions of the small intestine. In disease of the large intestines this attempt is absolutely unpromising.

**Prophylaxis.** In order to prevent the ingestion of the larvæ of sclerostoma horses should be prevented from drinking dirty water from pools or marshes, and they should be kept away from wet and marshy pastures. During stable-feeding care must be taken so that the drinking water is pure and free from contamination with offal and feces. Sometimes water has to be made safe by filtering or boiling. Mickley has succeeded in the Beberbeck stud, where formerly many foals succumbed to the disease, in eradicating it entirely since 1899 by the interposition of filtering boxes into the system of pipes of the water works. In studs or in large stables where many horses are kept and where sclerostoma infection is frequent, all animals should be examined according to the suggestion of Albrecht for the presence of sclerostoma and those found affected should be separated and kept away from the common pastures or exercise ground until free from parasites. Removal of the manure and frequent changes of the bedding straw are also indicated since the floor and the walls of the stables where horses are kept frequently come in contact with their feces and since they therefore become contaminated with sclerostomata and their larvæ, they ought to be carefully cleaned and disinfected from time to time. Marshy rough feed from marshy pastures should not be fed to horses. French veterinarians consider alfalfa and clover hay as dangerous in this respect.

Whether the intravenous injection of atoxyl (100 gm. of a 3% solution), recommended by Dorn, is indeed valuable in

eradicating sclerostomiasis in horses, has not yet been conclusively demonstrated.

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**Thrombosis of the arteria coeliaca.** Thrombosis of the arteria coeliaca (see page 395), which is extremely rare, like thrombosis of the anterior mesenteric artery, does not usually produce any disturbance in general health. A case described by Friedberger (Z. f. pr. Vet.-Wiss. 1875, 258) shows, however, that such a thrombosis may also cause symptoms of disease. The celiac artery in the case referred to contained two half cylindrical thrombi 17 and 25 mm. wide, the central end of the thicker thrombus was free in the trunk of the art. coeliaca. After motion the horse presented symptoms of severe colic and from time to time made peculiar noises such as are heard when horses are vomiting; it was also attacked at times by tonic convulsions. These attacks could be produced at will whenever the animal was made to move. Eleven days after the last attack the animal was killed and the postmortem examination showed a brown red discoloration of the gastric mucosa at the pyloric portion and signs of older hemorrhages in it, strong injection of blood vessels of the submucosa and hyperemia and hemorrhages in the first portion of the duodenum. The symptoms observed during the disease of the animal were believed to be due to a simultaneous aneurysm of the abdominal aorta, causing hyperemia of the brain or an accumulation of carbon-dioxide in the central nervous system; however, the symptoms were so similar to those observed by Marek on experiment dogs after ligation or obstruction of the art. coeliaca or some of its branches, that they may easily be associated with thrombosis of the celiac artery as it existed in this horse.

**Thrombosis of Mesenteric Veins.** Thrombosis of branches of the portal vein or of this vein itself has repeatedly been seen in domestic animals. Thrombosis of the portal vein is not uncommon in cattle and is caused by pressure of tuberculous masses or it may be due to injury by foreign bodies. Göhre and Spann have likewise observed thrombosis of the portal vein in cattle. Goubeaux, Collin, Mollereau, Cabaret, Kitt, Gratia have seen thrombosis of the portal vein of the horse; Connochie, a thrombosis of the anterior mesenteric vein; Siedamgrotzky, a thrombosis of both veins of the colon; in a case of Barrier there was compression of the portal vein by a carcinoma of the stomach. The authors saw a case in a dog where the large veins of the mesentery were compressed by a tumor of the mesentery. Thrombosis of the mesenteric veins is not rare in deeper inflammations or ulcerations in the intestines (Kitt) and it is possible that coagulation of the blood in the veins may occasionally be caused by thrombosis of the mesenteric arteries.

Symptoms of disease are produced only by thrombosis or compres-



sion of the larger venous trunks, while thrombosis of smaller individual branches, or an incomplete closure of larger veins, remains unrecognized. The mesenteric veins show anatomical conditions similar to those of the mesenteric arteries, but they are much more easily dilated, hence the conditions for establishing a collateral circulation in thrombosis of the portal vein are quite favorable. Siedamgrotzky, Mollereau and Connochie saw attacks of colic in the horse, Göhre in cattle; the attacks extended over a period of three weeks in Siedamgrotzky's case. Cattle show under these circumstances signs of a grave indigestion in addition or to the exclusion of other symptoms (Göhre, Spann). The authors have seen diarrhea and hemorrhages from the intestines in a dog due to compression of several veins of the small intestines.

Postmortem examination either shows abdominal ascites or a hemorrhagic infiltration of the intestinal wall. Compression of the trunk of the portal vein not infrequently causes chronic interstitial hepatitis.

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## 24. Internal Strangulation of the Intestine. Incarceratio et strangulatio intestini.

(*Darmeinklemmung, Darmverschnürung, Darmeinschnürung* [German].)

Internal strangulation of the intestines consists in a rapid closure of the lumen of the intestine by pressure from some body situated outside of the bowels, so that strangulated or incarcerated portion of the intestine at once shows signs of stagnation of the blood.

**Occurrence.** Horses are more frequently affected by internal strangulation than other domestic animals with the exception of oxen in some regions where they suffer frequently from so-called "spermatic duct strangulation" (see below under "Etiology"). Of the cases of colic in horses about 1 to 1.5 per cent are due to internal intestinal strangulation and of the fatal cases of colic about 5 to 13 per cent. Aside from the cases in oxen referred to above this form of displacement of the intestines is rare in cattle and other domestic animals.

Statistics of the Prussian army for the years 1892-1908 show that of 71,532 cases of colic among horses 1,069 cases (1.48%) were due to internal strangulation, and among these postmortem examination showed 304 (0.42%) to be diaphragmatic hernia; 234 (33%) displacements through the foramen of Winslow; and during 1892-1906 there occurred 218 cases (0.35%) of strangulation through clefts in the mesentery; 263 cases (0.43%) of strangulation by ligaments, pediculated tumors, etc. Among 8,426 cases of death from colic, 12.7% were due to internal strangulation.

In the Berlin Clinic there were, among 8,686 cases of colic which occurred from 1897-1907 ninety-three cases (1.1%) of internal strangulation, and among these thirty-four cases (0.39%) of strangulation in clefts or internal hernia; thirty-nine cases (0.45%) of strangulation by ligaments, etc.; seventeen cases (0.2%) of dis-

placement through the foramen of Winslow. Among 1,408 horses dead from colic, 6.6% had suffered from internal strangulation.

The statistics of 1889-1894 and 1896-1908 of the Dresden Clinic show among 3,336 cases of colic, thirty-two cases (0.9%) of internal strangulation; among these four (0.12%) of diaphragmatic hernia; twenty-three (0.69%) of strangulation by ligaments, clefts, etc.; five (0.15%) of displacement through the foramen of Winslow. Among 500 horses dead from colic, 6.4% died of internal strangulation.

In the Budapest Clinic there were seen from 1900 to 1909 among 5,487 cases of colic, fifty (0.9%) due to internal strangulation. Among 725 horses dead from colic, 6.9% were due to internal strangulation.

From the above data it appears that internal strangulation is comparatively frequent among cavalry horses and that among these cases those due to hernia of the diaphragm, to displacement into the foramen of Winslow and into clefts formed previously to the strangulation, are more common than they are among other horses.

**Etiology.** The intestines may become strangulated by various strands and bands present in the abdominal cavity. In this respect are to be mentioned congenital diverticula of the ileum (Meckels diverticula); exceptionally also acquired diverticula of the intestines (Kitt); the ligament between kidney and spleen (Marek, Forssell), the spleno-gastric ligament, the falciform ligament of the liver (Blanc), the urachus, the small omentum (Dupuy); pediculated neoplasms (generally lipomata) arising from the abdominal wall or the mesentery, possibly also from other abdominal organs, or exceptionally pediculated lobes of the liver (Pécus); in horses also exceptionally a stump of the spermatic cord, which has slipped back into the abdominal cavity, or the spermatic cord of an undescended testicle (Gutbrod); the larger omentum adherent to the anterior abdominal wall and twisted into a cord, sometimes the anterior mesentery itself (in a case of Dupuy and one of Prince, the cecum became strangulated); finally postperitonitic connective tissue bands with one free end or with ends connecting various abdominal organs. *Cysticercus tenuicollis* caused strangulation of the duodenum in a hog (Späthe).

In other cases internal strangulation is brought about by portions of the intestines getting into congenital or acquired openings of the mesentery or of the omentum, or into torn and partially detached portions of the peritoneum or into a cleft between separated muscles (Roy). Some of the above mentioned ligaments may also tear and in this manner produce intestinal strangulation.

In a similar manner there develops in oxen or young castrated cattle, what is called internal hernia (*Strangulatio ducto-spermatICA*) a condition studied by a number of veterinarians, recently especially by Walch. This form of strangulation occurs only after castration with twisting or rather tearing off of the testicle, especially if the animals are castrated during the first weeks of life. Walch has also shown experimentally, that in such cases the reduplication of the peritoneum (pouch of Douglas) attached to the spermatic cord between the internal opening and the seminal vesicles, usually tears, so that loops of intestines slip into the opening so made, either immediately or later on. In other cases the stump of the spermatic



cord retracts after tearing off its peritoneal covering, and gets from the spermatic canal into the abdominal cavity, or the spermatic cord is torn in its abdominal portion. In either case the remnants of the cord will either float as a free stump in the abdominal cavity or it will become adherent to the peritoneum and form a cleft. Since castration is performed according to different methods in various places, the affection only occurs in such regions where castration is performed as above described. Walch found this abnormality of the spermatic cord without strangulation in oxen in 60 to 70% of the slaughtered animals. Tearing of the fibrous tunic of the external inguinal ring by a strong pull can also be detrimental if the spermatic cord is severed transversely, and if the testicle and vessels are then pulled off. A case of strangulatio ducto-spermatica in a horse has been described by Janson; in this case the pouch of Douglas had likewise been torn. Metzger and Strauss have exceptionally seen the affection in sheep. Tearing of the reduplication of the peritoneum in sheep and in other animals is rare, since this fold of peritoneum is more resistant and smaller than in cattle.

So-called internal-hernia will lead to intestinal strangulation, either from the start or later on. Diaphragmatic hernia occurs particularly in the horse, more rarely in cattle and dogs. It is either congenital or formed during extrauterine life, the diaphragm rupturing in consequence of too great, sudden intra-abdominal pressure, or the accident may occasionally be due to dilatation of the stomach or to bloating. Such ruptures are most common in horses which have to jump (hence they are most frequently found in army horses). Displacement of the intestines into the foramen of Winslow occurs exclusively in the horse and the intestine then gets between the layers of the great omentum (enterocele omentalis). (What Lucet & B have described as an enterocele omentalis in cattle, was, according to Mathis, only a strangulation into a tear of the omentum.)

Incarceration of external hernia, which is a surgical lesion, leads to the same local symptoms and to the same clinical picture as internal strangulation.

Among the enumerated causative factors are some (incarceration in narrow clefts) which are able to cause strangulation at once, but additional predisposing causes are, as a rule, necessary to bring this condition about. A slight filling of the intestines enables loops to slip into a preformed opening. In this condition the intestines can also easily change their position, and openings usually covered by the large intestine (foramen of Winslow, tears in the lower portion of the diaphragm) can now become accessible to the small intestines. Active peristalsis, whether it be due to external or internal cooling, tarrhal affections, embolism, etc., strong action of the abdominal press (pulling of a great weight, galloping, jumping, walking uphill, efforts, rolling, falls, being run over), favor the displacement.



ment of loops of intestines into a cleft during energetic movements of the body, or pediculated formations or bands which are free at one end may in rolling become twisted around loops of intestines. Under similar conditions strangulation is produced by the spleno-nephritic ligament, a variety of strangulation which is not rare. (In five cases observed by the authors the left portion of the colon was always strangulated.)

The jejunum, which is most motile and most slender, is strangulated most frequently; other portions of the intestines are less commonly affected in this manner.

**Pathogenesis.** If the opening into which a portion of intestines has become displaced is narrow compared with the caliber of this part of the bowel, or if a band-like body has been twisted around the intestines, the intestinal wall and its mesentery are compressed to a certain degree and the lumen becomes closed. In other cases the pressure becomes effective only if the other additional factors mentioned above have caused further portions of intestines and mesentery to become displaced, or after the portion of the intestine that was first displaced has become bloated in consequence of kinking or has become swollen in consequence of venous (passive) congestion.

The outflow of venous blood from the affected intestine and its mesentery is interfered with in proportion to the pressure exerted, while the thick-walled arteries with their high internal pressure keep on pumping blood into the tissues, possibly in a somewhat decreased amount. Hence, stagnation of blood (stasis) soon occurs in the incarcerated portion of intestines and also, owing to an increase in venous pressure, a diminution of the velocity of the entire blood current with all the consequences which have been discussed in detail under the head of thrombosis of the mesenteric arteries (see page 396). At the place of strangulation the compression produces an anemic ring.

The rapidly increasing stasis, and the overloading with carbon-dioxide due to it, excite strong, frequent and occasionally convulsive contractions in the strangulated portion of intestines, causing colicky pains. The frequency and the intensity of these convulsive contractions may be the greater, the longer is the portion of intestine which is strangulated. A stronger pressure, which, however, does not completely shut off the arterial blood supply, clearly causes a considerable overloading of the blood of the affected portion with carbon-dioxide, and causes stronger contractions. Everything else being equal, strangulation of the small intestines which are rich in nerves causes more intense abdominal pain than strangulation of other portions.

Strong contractions may cause the entrance of more loops through the place of strangulation by suction or traction if the part situated towards the anus becomes distended, so that it pulls on the part situated more towards the stomach (Wilms).

Throwing down or rolling with suddenly increasing abdominal pressure may, on the other hand, force more loops of intestines through the place of strangulation by pressure from behind (Kertész).

The strangulated portion of bowel, which is filled with intestinal contents mixed with extravasated blood, will soon become bloated, because the gases can neither be removed towards the stomach nor towards the anus, nor can they be absorbed by the blood. Peristalsis and pain finally cease in the strangulated portion after it has been dilated ad maximum and after edematous infiltration has occurred. Peristalsis of the portions situated between the strangulation and the anus cease almost at once or shortly after the strangulation occurred. Contractions persist for a long time in the portions situated anteriorly to the point of strangulation, and some may later become convulsive and cause pain; the latter is, however, not very severe, as shown by the experiments of the authors. Peristalsis in the last mentioned portion of the bowel decreases as the intestinal contents undergo a more and more active fermentation, and finally they likewise cease.

The gradually increasing swelling of the intestinal wall and of the mesentery, and the involvement of more loops of intestines produce increased pressure upon the sensory nerves that are caught in the place of strangulation; this causes a continuous, but not severe pain, as experiments on horses show; the latter may even be entirely absent where the strangulation is not too severe. In the further course continuous pain, which increases on pressure, is also caused by peritonitis which develops. The general symptoms are similar in origin and character to those encountered in thrombosis of the mesenteric arteries (see page 403); however, they vary in different species of animals as to their appearance, according to variability in susceptibility to microorganisms and their toxins. The shorter the strangulated portion of intestine and the less firm the strangulation, the later and the less severely will these general symptoms appear.

**Anatomical Changes.** The strangulated portion of intestine appears, as a rule, dark to blackish red, distended and tense, the wall thickened by serous-hemorrhagic infiltration, friable, the intestinal lumen filled with a thin fluid, fetid, hemorrhagic mass, the mucosa blackish red, on the surface dirty yellowish, smeary. The site of the strangulation is indicated by a pale yellowish strip; sometimes the strangulated portion shows several segments, separated from each other by pale rings with alternating reddened portions (gradual entrance of the strangulated portion in sections). Aside from strangulation a circumscribed or diffuse peritonitis, and perhaps intestinal rupture may be found. The peritoneal cavity contains a yellowish or reddish serous exudate, frequently mixed with shreds of fibrin.



**Symptoms.** In horses, sudden, rarely gradually developing symptoms of violent abdominal pain are observed; in some cases the animals previously show the symptoms of stenosis of the intestine. Usually the animals throw themselves down recklessly, roll, kick with their feet, and their restlessness may even assume the character of a maniacal excitement. Later they avoid reckless throwing; they lie down, but do so carefully, or they stand still, probably in consequence of the development of peritonitis, which causes pain. Rarely do the animals behave quietly and with care from the start, and do not throw themselves down, and in such cases one might think of a stronger constriction of intestinal and mesenteric nerves at the place of strangulation. Sometimes a reckless behavior persists until the end. Stretching, lying down on one side, squatting dog fashion on the haunches, kneeling, lying on the back are observed frequently; these positions have, however, no particular significance. The restlessness may be almost absolutely continuous, so that the patients are still hardly a moment, either in standing or in the recumbent posture, or the restlessness comes on in short attacks; this depends upon the intervals at which the convulsive intestinal contractions occur. In strangulation of the rectum or small colon, strong pressing down is seen occasionally. The expression of the face betrays distressing pain the same as in intestinal displacements in general.

The abdominal circumference is rarely increased, and if at all only moderately so. Intestinal sounds may be increased for some time in strangulation of the rectum or small colon; in

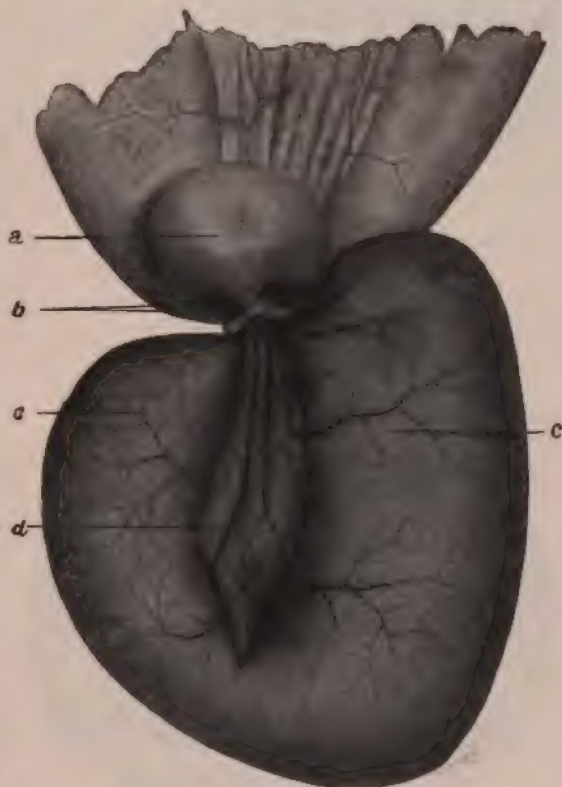


Fig. 44. Loop of small intestine strangulated by a lipoma: (a), lipoma. (b), its pedicle, which forms a ring. (c), strangulated loop of intestine with swollen and much congested mesentery. (d).



other cases they soon become less frequent and are eventually suppressed.

Defecation ceases at once or soon; in exceptional cases, when the strangulation is not complete, there may be only retarded defecation in the further course of the disease.

Rectal examination shows that the rectum is empty and frequently the strangulation of some parts of the intestines, its seat and its origin, can be ascertained. In strangulation of the rectum the hand meets an impediment and it is frequently impossible to pass even one finger. The wall of the intestine is folded in front of the obstruction and is tender; if it is possible to press the finger through the impediment this is found coated with a dirty, reddish, fetid fluid. In strangulation of any other portion of the intestines one can ascertain that there is

somewhere a much bloated intestinal portion which decreases in one place to a folded string; here the tissues are tender and surrounded by a tense ring. Sometimes one finds pediculated tumor-like formations, and occasionally it is even possible to determine the direction in which the constricting band or the pediculated body has become wound around the bowel. If the place of strangulation is in the anterior portion of the abdomen, which cannot be reached by the hand,



Fig. 45. Loop of small intestine (*a, b*), strangulated in a cleft of the mesentery (*c*).

it is possible at most to demonstrate circumscribed bloating and tenderness of certain portions of the intestines, or the examination may at first be perfectly negative while later on a gradually increasing bloating of moderate degree will be noticed of the parts situated in front of the strangulation towards the stomach.

In strangulation of the intestines by the nephro-splenic ligament, one can ascertain that the spleen is not in contact with the left abdominal wall as usual, and its base has been displaced far downward, approximately to the middle of the left abdominal wall, while the ligament has been drawn out to a cord 15-20 cm. long. In the ring which is not closed entirely and is formed by the nephro-splenic ligament, the free upper end of the base of the spleen, which is usually horizontal, by the left kidney and the left abdominal wall, one finds the left portion of the colon suspended

on the nephro-splenic ligament; the upper left loop of the colon usually rests directly upon the ligament and appears strangulated, while the lower left loop which is now directed upward and to the left is not compressed or very little. The posterior half of the bloated left portion of the colon runs backward and downward in a curve, and the upper loop is more or less situated in front of the lower loop. If at the same time the pelvic flexure is directed forward and somewhat laterally, the bands of the lower portions present a spiral course. Strangulation of loops of small intestines, which the authors have seen occasionally in other forms of colic, was never found in this affection.

According to Larsen the presence of some tense loops of small intestines in the region of the right flank, directly at the abdominal wall, points to displacement of the small intestine through the foramen of Winslow; this was also found in a case of Forsell. The authors have had similar findings in displacement of the small intestines, due to dilatation of the stomach.

In diaphragmatic hernia respiration is difficult from the start on walking downhill, and in certain positions on the side. Over the posterior and lower portions of the thorax one finds tympanitic and often metallic sounds, which frequently change in pitch. Soon there appear symptoms pointing to acute pleuritis (tenderness on pressure upon the intercostal spaces, friction sounds), or to accumulation of blood in the pleural cavity. In diaphragmatic hernia of the left side the heart may become displaced by prolapsed loops of intestines and the apex beat may disappear.

A peculiar type of dyspnea was seen in a horse where a recent extensive rupture of the diaphragm gave rise to a prolapse of the small intestines and the stomach. In spite of marked and rapid excursions of the ribs a passive drawing in of the intercostal spaces did not occur, but, on the contrary, a strong inspiratory drawing in of the epigastrium, the flanks and the region of the loins. The cause of this form of dyspnea was found in the large extent of the rupture of the diaphragm; during each inspiration a part of the small intestine was drawn through the opening of the diaphragm into the thorax and the intraabdominal pressure was diminished in consequence.

In strangulation of the small intestine acute dilatation of the stomach (see page 298) is frequently seen later on.

The behavior of the pulse, sensorium, temperature and irritation are similar to what is seen in grave forms of emphysema and thrombosis (see page 406); the pulse rises after a few quarters of an hour, or at least after a few hours, to above sixty per minute, and as it grows faster it grows weaker, the sensorium becomes clouded, respiration difficult and the temperature rises.

In strangulation of a very short piece of intestine, it may occur, however, that acceleration of the pulse and elevation of temperature takes place very slowly and does not reach a high degree. In a horse where a piece of small intestine 30 cm. long became strangulated by a pediculated lipoma, the pulse was only 56 after 13 hours and the temperature 38.6%, yet, when a laparotomy was made 2 hours later, fetid fluid was found in the abdominal cavity and necrosis of the intestinal wall.

The clinical picture in cattle is also characterized by marked symptoms of colic. The animals kick with their hind legs and push against the abdomen with their horns; they shake their heads, look around towards the abdomen, lie down, soon



get up again, trip restlessly from place to place and sometimes step into the crib. The gait is sometimes stiff, because the right hind foot is not placed sufficiently forward. The restlessness ceases after several, usually after 6 to 12 hours; it may, therefore, happen that, if the attack occurred in the evening, it is over the next morning, and that only the condition of the bedding straw shows that the animal has been restless during the night (Walch). Symptoms of restlessness are absent during the further course, and with the deterioration of the general condition one observes only groaning and slight restlessness at longer intervals, due to the peritonitis which has developed. Perspiration is frequent at the onset of the disease.

The circumference of the abdomen is at first not changed; later on moderate bloating occurs, due to moderate dilatation of the portions of intestines nearer to the stomach, and also on account of secondary bloating in the rumen. The intestinal sounds are suppressed and cease entirely later on; sudden pressure on the right half of the abdomen later produces splashing sounds and pressure upon the abdominal wall, especially on the right flank, often produces symptoms of pain. After restlessness has set in the feces are voided for a short time, for a few hours at the utmost, and afterwards complete constipation supervenes; perhaps very little fecal matter or some mucus may be expelled with great efforts.

Rectal examination, which, according to Walch, is best made on the standing animal, will always (Walch) reveal the spermatic-duct type of strangulation (*strangulatio ducto-spermatica*), and frequently also the other types. The rectum is found empty. In strangulation of the spermatic duct type, one finds a tumor, the size of a fist up to the size of a child's head, occasionally as big as an adult human head, in the neighborhood of the internal inguinal ring, as a rule on the right, very rarely on the left side. The tumor is composed of tense dilated intestinal loops and is adherent at one point to the abdominal wall, where one can feel a tense cord. If the retracted spermatic cord has become wound around the intestines, the tumor is more motile and is nearer to the median line or in the middle of the anterior margin of the pelvis; in this case one can also feel the strangulating cord. In strangulations of other types the findings in cattle are the same as they are in horses.

In spite of the fact that the animal has subsequently quieted down, the sensorium becomes clouded. There is complete lack of appetite and great weakness, so that the animals are no longer able to get up.

The pulse early becomes rapid and weak, and it rises to 120 to 130 per minute; simultaneously with this acceleration, the peripheral portions of the body become cool, the visible mucosa, which is at first reddened, becomes pale. The respiration is accelerated from the start and becomes more difficult.



The elevation of temperature which sets in later, announces the development of peritonitis or of general infection.

**Dogs** become depressed, they lie around a good deal, sometimes they cry out, groan, whine, constantly change their place, sometimes look around towards the abdomen, get up suddenly, roll, stretch their extremities stiffly, or remain for a long time resting on their abdomen. They frequently exhibit an irritable temper. Gagging or obstinate vomiting are seen frequently. Constipation is complete from the start.

The abdominal circumference is at first normal, it may become larger in the further course; the abdominal walls are tense, and strong pressure at certain points excites pain. In palpation of the abdomen, which must be made under a slight narcosis if there is great tension, one can feel somewhere in the abdominal cavity, distended painful loops of intestines; sometimes the strangulation can be mapped out as a firm, stretched painful mass.

The restlessness, which is not very great, decreases within the next hours, but the deterioration of the condition is shown by increasing depression, weakness, elevation of temperature, a weak and rapid pulse, continued-obstinate vomiting and constipation. As in serious diseases of dogs in general, one may in this case observe a lowering instead of an elevation of temperature.

In **hogs** Späthe described a clinical picture in intestinal strangulation similar to that observed in dogs.

**Course.** The disease is characterized in all species of animals by a sudden onset; sometimes it follows upon a manifest predisposing cause, which is preceded in infrequent cases by the symptoms of intestinal stenosis. Pain is either intense from the very onset or it may increase gradually, but rapidly; this depends upon whether the strangulation has been produced suddenly or has become complete only after some time. Severe pain persists as long as the incarcerated intestines retain their power to contract; according to variability in the degree of strangulation this may last from a few hours to one day. Later on the animals become apparently perfectly quiet or only occasionally exhibit some signs of abdominal pains which are caused by contractions of the intestines situated between the place of strangulation and the stomach, or by peritonitis which has set in in the meantime.

If the animals are left to themselves they soon perish in consequence of rupture of the intestines, general sepsis or infection. Horses usually, dogs frequently, succumb during the day while cattle remain alive for two to six days (Walch).

The possibility in the beginning of the affection of a spontaneous reduction of the strangulated portion of intestines must be admitted in exceptional cases.

**Diagnosis.** Internal strangulation of the intestine can be diagnosed with certainty only if rectal examination or palpation of the abdomen furnish a positive result; all other symptoms can at best furnish a more or less well founded suspicion. If there is good ground for a strong suspicion an exploratory laparotomy should be made in cattle and in dogs, occasionally also in other animals; this enables us to determine, by the aid of the hand which is introduced through the laparotomy incision, the seat and origin of an existing strangulation. In steers less than 9 to 10 months old, where a rectal examination cannot be made, an exploratory laparotomy should be made in all cases when sudden symptoms of colic have come on with constipation, and where treatment during a period of twelve hours has not brought about any improvement (Hoffmann). If there are symptoms pointing to obstruction of the intestines and one hears over the posterior, lower parts of one side of the thorax tympanitic or possibly metallic percussion sounds which are variable in pitch and intensity, if quite intense intestinal sounds are audible in this region, and if symptoms of beginning pleuritis or hemothorax appear soon, then a diagnosis of incarcerated diaphragmatic hernia appears safe, especially after an exploratory puncture of the thorax has furnished hemorrhagic intestinal contents. Aside from strangulation, circumscribed intestinal meteorism is encountered also in torsion of the intestine, in the grave form of thrombosis, in acute dilatation of the stomach, hence this can only be utilized as a suggestive symptom in connection with other symptoms, and with certain circumstantial evidence in those cases where the exact site of the incarceration cannot be felt or otherwise ascertained directly. However, if dealing with a strong meteorism localized to a few loops of intestines, one is seldom in error in thinking of intestinal strangulation, torsion or volvulus. The clear, yellowish or slightly reddish serous fluid, which is at first obtained by exploratory puncture of the abdominal cavity, is found in internal strangulation, torsion, invagination, and in the graver forms of thrombosis of the mesenteric arteries.

The differential diagnosis has to consider in particular acute dilatation of the stomach, the grave forms of thrombosis, torsion, volvulus and invagination of the intestine, incarcerated genuine hernia, internal obstruction of the bowel and in cattle also obstruction of the urethra by calculi.

Acute primary dilatation of the stomach of the horse can be excluded with certainty, in the majority of cases, by anamnestic data, such as early difficulty in respiration, absence of early and marked disturbances of defecation and rapid improvement, and definite recovery after the proper use of the stomach tube. Torsion, volvulus and invagination can only be differentiated from internal strangulation by the findings on rectal examination. It is not of very great importance if it is impossible

to differentiate the various forms of intestinal displacement, since the treatment is the same in all three forms. Incarcerated external hernia may be recognized on the basis of the symptoms of closure of the intestines in connection with the local signs; hence an examination of the inguinal ring and of the scrotum should always be made whenever there are colicky pains, particularly in stallions. Changes of position and internal strangulation may be confounded with those types of intestinal obturation (impaction) which cause almost continual pain and tenderness upon pressure in well defined places of the abdominal cavity (see page 382). Similar symptoms have occasionally been observed in cattle in obturation of the bowel by blood coagula. Aside from the result of rectal exploration, the continuous absence of general symptoms and anamnestic points frequently furnish sufficient diagnostic data. Obstruction of the urethra by calculi in male cattle is distinguished from intestinal strangulation by the following features: Urination ceases in obstruction of the urethra, but not the defecation which goes on undisturbed, the patients exhibit peculiar pulsating motions of the urethra in the region of the intestines, rectal exploration in the inguinal regions is negative and one can only feel the urinary bladder in the shape of a flat cylindrical, thick tumor, lying exactly in the median line.

**Prognosis.** The prognosis is unfavorable if rectal or operative correction of the intestinal strangulation can, for some reason or other, not be accomplished, because spontaneous recovery occurs only very exceptionally if at all, and medicinal treatment has no influence whatever upon the affection. The operative procedure is determined preferably by the species of the patient, also by the duration of the train of symptoms, the general condition of the animal, the condition of the constricting body, and by the question whether reposition can be accomplished per rectum or only by a laparotomy. The prognosis is most favorable in cattle, even in such cases where a laparotomy is necessary. (Hoffmann observed losses only in 5% out of 185 cases.)

Dogs likewise stand the operation of laparotomy well. The presence of a secondary peritonitis or of grave disturbances of the general condition very much diminish the chances of operative procedure, and these are abolished completely if symptoms of collapse (unconsciousness, pale mucosæ, subnormal temperature, unrecognizable pulse, paralytic weakness) and high fever in horses are already present. Exploratory puncture may furnish certain data of prognostic value; if a putrid smelling fluid is obtained from the abdominal cavity there is no more hope of saving the animal.

**Treatment.** The nature and the manner of the origin of the affection determine the fact that only operative procedure



can accomplish something; the object of the operation is to relieve the strangulated intestine; this may be accomplished from the rectum in some cases, in horses as well as in cattle. However, a laparotomy cannot be avoided in the majority of cases in the horse and frequently also in cattle, and this operation is the only one possible in smaller animals.

One cannot lay down a uniform law for the reduction per rectum, since local conditions vary not only as to different, but even as to the same types of intestinal internal strangulation. However, the more frequently rectal exploration is practiced in the examination of patients, the more frequently are found cases in which it is possible to liberate from the rectum loops of intestines that are strangulated by bands or pediculated tumor, either by untwisting a band or by tearing it or the slender pedicle of the tumor. Perl succeeded in tearing with his spread fingers the pedicle of a lipoma, the pedicle having strangulated the rectum (the authors attempted the same procedure in two cases of strangulation by pediculated lipomata, but they were not successful).

Strangulation of the intestines by the nephro-splenic ligament may be overcome in horses in the following manner: The right hand is introduced into the rectum of the standing animal, and it is then pushed between the stretched nephro-splenic ligament and the compressed bowel; the latter is then grasped to the right and below and is pushed towards the left, being at the same time pressed down with the thumb. While executing this rotating motion towards the left, the back of the hand attempts to displace the base of the spleen towards the median line of the abdominal cavity. The intestines can usually be released within fifteen or thirty minutes, occasionally within a few minutes. In four of the authors' five cases, they succeeded without trouble, but in one case a laparotomy became necessary. Forsell has relieved cases of this type by this method; he placed the patients on the back.

In spermatic duct strangulation of oxen one may, if the condition has not lasted long, attempt to press the intestines which are not yet bloated through the strangulating ring. As a rule, however, the animals are only seen after they have been sick some time, then the spermatic cord must be separated or torn loose. After the posterior portion of the abdomen of the patient has been elevated, the well-greased arm is introduced into the rectum up to the elbow, then one attempts either to separate the duct or to tear it loose. The operative procedure is variously described by different authors. Walch grasps the cord between thumb and index finger, then closes the hand and turns it downward and to the right, and also draws it backwards and to the left.

Servatius grasps the cord from above, from right to left and winds it once around the index finger in a spiral; he then closes this finger and presses it against the thumb. Then he pulls towards the abdomen and tears the cord away from its attachment. In animals more than two years old, when the attachment of the spermatic cord is much firmer, he attempts to grasp it with the whole hand, brings it to the back of the hand, winds it around the wrist and now tries to tear it loose with one strong pull; if possible an assistant pulls at the same time on the scrotum.

Hoffmann draws the cord with the tip of the fingers or with the whole fist from in front upwards and then from within backwards into the middle of the pelvis; the cord is usually torn during this manipulation. If, however, a tear does not occur, he winds the elastic cord from one to three times around his hand and changes the direction of the traction backwards, upwards and to the left.

The described procedures usually succeed in relieving internal strangulation in oxen. (Probst succeeded in twenty-seven case out of twenty-nine.)

Such procedures as driving uphill, elevating by pulling high the hind portion of the animal, rolling on the back, are absolutely unpromising in internal spermatic duct strangulation. The proprietors of animals must be taught to avoid methods of castrating male calves which are liable to bring about this affection.

In smaller animals or in calves less than 9 to 10 months old, laparotomy becomes necessary if the animals are to be saved. This operation likewise is usually necessary in horses, and also in cattle in all other forms of strangulation except spermatic-duct strangulation; in oxen older than one year, the latter rarely necessitates laparotomy. It is obvious that one must not wait with laparotomy until necrosis of the intestinal wall or a diffuse peritonitis have developed. The abdominal incision is to be made as near as possible to the location of the strangulation; the strangulating band must be severed, but clefts should be enlarged by a bloodless method, that is, by stretching or tearing. In this manner recovery has frequently been brought about in internal hernia of cattle and occasionally, also in other types of strangulation of this species (Kränzle, Schiel).

Plósz & Marek and also Teltsch have performed laparotomy in the horse, each in one case of intestinal strangulation; the band was severed with the aid of a pair of scissors introduced into the abdominal cavity. Rogerson, however, did not succeed in relieving a strangulation caused by a tumor. Forsell made an interesting report of the details of three cases of laparotomy performed in horses for the relief of strangulation of the intestines through the foramen of Winslow. One of the cases terminated in recovery, two of the horses died, one of them from hemorrhage due to injury of the portal vein.

Laparotomy is, however, not always indicated, even in cases where a reliable diagnosis has been made; the very nature of the affection in some types excludes the possibility of removing the cause.

Laxatives can only do harm in strangulation, they must, therefore, be avoided. Narcotics are indicated for easing the pain, vasotonics to increase blood pressure.

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**Incarcerated genuine hernias** produce the same general clinical picture as internal strangulations of the intestine; their treatment rests upon the same principles.

## 25. Volvulus and Torsion of the Intestine. Volvulus et torsio intestini.

Volvulus and torsion of the intestine is a rapid closure of the bowel lumen, caused by a turn of the intestine around its own axis or by strangulation caused by other loops; simul-



taneously with it stagnation of the blood occurs in the affected portion.

On the basis of this definition postmortem torsion, which is comparatively frequent, must be separated from volvulus and torsion; and also displacements of the intestine which are not really pathologic in character and occur quite commonly when certain portions of the intestine are distended with masses of gas or feces, which are, however, not accompanied by an encroachment upon the intestinal lumen, nor by a stagnation of blood in the intestinal wall.

**Occurrence.** Volvulus and torsion of the intestine occur with rare exception only in horses. About 2-5.5% of the colics of the stomach and intestines are due to this cause. Oppenheim, Reichert, v. Ow, Guittard and Joehnk have each seen one case in cattle; Ries two cases; Hess saw torsion of the cecum in a calf four days old. Glage saw a few cases of torsion of the cecum in hogs. Liénaux and Probst each saw a case of volvulus in a dog; Johné a case in an elephant.

Volvulus and torsion in horses are not seen everywhere equally often. This depends upon a difference of susceptibility in various breeds and on differences in the attendance and in the use of horses in different countries. Torsion of the cecum is most frequent in countries and states where the heaviest horses are kept (the affection is more frequent in South Germany than in North Germany). An increase of the number of cases of torsion of the cecum has been noticed more recently and is probably due to the fact that more attention has recently been directed towards torsion of the cecum, which is not always easy to detect even anatomically.

The disease is seen comparatively frequently in army horses. Between 1892-1908 the horses of the Prussian army furnished 71,532 cases of colic, among which were 2,893 cases (4%) of volvulus and torsion; 1,585 cases (1.93%) of volvulus with rare examples of torsion of the small intestines, or of volvulus or torsion of the small colon; 1,532 cases (2.14%) of torsion of the colon; ninety-two cases (0.12%) of torsion of the cecum. In 8,426 cases of death from colic this form of intestinal displacement was represented in 34.5%; among these volvulus and torsion of the small intestines in 16.1%, torsion of the colon in 18.2%.

In the Berlin Clinic there were 8,686 cases of colic (1897-1907), and 437 of these (5%) were due to volvulus or torsion; volvulus or torsion of the small intestines or of the small colon occurred 183 times (2.1%); torsion of the large colon 242 times (2.8%) (in four cases of the colonic flexure); and there was one case of torsion of the cecum. In 1,408 fatal cases, displacement of the small intestine and small colon was represented with 13%, and displacement of the colon with 17.1%.

During the years 1889-1894 and 1896-1908, 3,336 cases of colic were seen at the Dresden Clinic; among these were 183 cases (5.5%) of volvulus and torsion; sixty-five cases (1.95%) were volvulus of the small intestines; 117 cases (3.5%) torsion of the colon and one case (0.03%) torsion of the cecum; 36.6% of the deaths from colic were due to this form of intestinal displacement and among these 23.4% to torsion of the colon and 13% to volvulus of the small intestine.

From 1900-1909, 5,487 cases of colic were seen in the Budapest clinic; among them 101 cases (1.8%) of volvulus and torsion, viz., fifty-four cases (0.98%) of volvulus or torsion of the small intestine; forty-four cases (0.03%) of torsion of the cecum, and one case (0.01%) of torsion of the small colon; of the fatal cases 13.9% were due to volvulus or torsion of the intestines.

**Etiology.** Volvulus and torsion of the intestines are without doubt brought about in a majority of cases by mechanical causes. Experimental proof of this statement has been furnished by observations that animals acquired this intestinal displacement immediately after certain mechanical injuries, and died from it (Mayer, Glage, Liénaux), or that intestinal torsion



occurred secondarily, in consequence of rolling in the course of other painful intestinal affections or displacements (strangulation, invagination), (Rulf, Jöhnk, authors' observation). The importance of mechanical factors as a causative agent is also proven by postmortem displacements which are seen particularly often if cadavers of horses have been transported before postmortem rigor had set in. Aside from rolling, abnormally intense peristalsis rarely plays a rôle in the production of displacements, and if at all, it does so preferably in displacements of the small intestine or small colon.

**Torsion of the left portion of the colon (torsio coli)** in horses may be brought about in a double manner. When the tolerably long left portions of the colon are well filled and the horse turns rapidly from one side to the other, it may occur that the left portions are not able to follow rapidly the quickly moving right portions. If now the horse, when lying on one side, turns to the other (Jelkmann), or rolls, no matter for what reason (colicky pains), or if it is thrown and turned (Mayer), then displacement may take place in the manner indicated. In such cases the left portions represent the fulcrum, around which the right portions turn, which are fastened to the upper abdominal wall.

In the majority of cases the divisions of the colon show an opposite behavior when torsion occurs. The right or the transverse divisions form the fulcrum, around which the remaining free divisions turn around their axis, either towards one side or the other. In reckless lying down or throwing down, in rolling, in rapid gait, or in jumping, that is, in all forms of motion where the abdomen makes an extensive turning or lateral movements, it may likewise occur that the free and quite heavy left divisions of the colon may make swinging or turning motions along the abdominal wall, either to the right or to the left, particularly if they are not well supported by other intestines. The upper division may sink down, especially in its terminal portion which is more or less filled with voluminous and firm contents, and pull the other divisions with it, either along the abdominal wall or along the median line of the inferior division. The originally not twisted portion will probably not sink into the torsion in addition to the other divisions, because the contractions of the two divisions occur in opposite directions, and so paralyze or neutralize each other.

Intense and convulsive peristaltic contractions (dysperistalsis), which play an important rôle, according to the belief of Forssell, cannot, directly, bring about torsion of the colon, in the opinion of the authors, because the connected portions of the colon must remain together even if the contractions are irregular. It could not be well explained how the contracting division could get beneath another, quiescent division or how the several positions of the colon could be displaced among each other in consequence of abnormally strong contractions. Convulsive contractions play a predisposing rôle in that they excite colicky pains and cause the animals to throw themselves or roll, and these movements then may cause torsion. In this respect disturbance of circulation caused by thrombosis of the mesenteric arteries, acts like other colicky affections with violent pain.

Predisposing causes always play an important part in connection with the immediate exciting causes. As such may be mentioned heavy filling of the left divisions, especially of the pelvic flexure, with fecal masses, dry masses of feces, sand or calculi, while the transverse divisions are filled less completely; also the stronger filling of the right upper division and, according to Forssell, a stronger filling of the left lower division, the presence of a tumor in the wall of the intestines; a minor degree of filling of the other portions of the bowel; also probably, the variable condition of the stomach as to a greater or a lesser degree of filling and a relaxation of the abdominal wall. Forssell also called attention to it that a predisposing cause is furnished in some horses by the comparatively large mesentery between the divisions at the pelvic flexure.

If the divisions of the colon are filled uniformly with firm feces or if they are bloated uniformly, torsion cannot occur. In such cases the internal abdominal pressure is much increased and the free movements of the intestines are inhibited proportionately; uniformly distributed voluminous contents of the bowel or a great tension of the wall do not permit a decrease of the intestinal lumen or a compression of the vessels, and these factors are necessary for the production of torsion. In cases where observers have believed torsion to have occurred in consequence of bloating, they were evidently mistaken; torsion occurred first and bloating later on as a consequence.

**Torsion of the cecum** around its long axis is very rare, and it is probably also caused by the mechanical influences pointed out above. The shortness of the cecum when compared with the colon and the position of the former, fully explain why displacements of the cecum should be so rare.

**Volvulus or torsion of the small intestine** or of the **small colon** have a much more complicated mechanism. Torsion may be brought about by a loop of intestine with its mesentery becoming twisted around another loop, the latter with its mesentery forming the axis (volvulus, formation of a nod, volvulus nodosus), or by a longer loop turning around its own mesentery (axial torsion, volvulus mesenterialis). In some cases the whole small intestine of the horse, or the whole colon in the hog may be twisted around the larger omentum with its mesentery, particularly if the body performs very abrupt motions, if the animal throws itself around or is rolled, as occurs frequently in the unloading of hogs and calves. The production of torsion is favored if the intestines contain an abundance of liquid or dry feces or sand (Glage), or if they are the seat of a tumor. However, total or even partial torsion of the mesentery is quite rare.

Especially in horses one observes more frequently entangling or volvulus of the small intestines alone; loops of small intestines are very rarely twisted around the colon or cecum or around the small colon, while loops of the small colon rarely become twisted themselves. In many of these cases, reckless, ex-

tensive motions, throwing down, rolling, jumping, etc., cause loops of intestine to become twisted around others.

One may also consider another mode of production. Especially in the horse, loops of small intestine often cross each other in several places under perfectly normal conditions; this arrangement becomes more marked in increased peristalsis, and loops of intestine with a longer mesentery can then move to distant places in the abdominal cavity and below the heavy loops of the large intestine. If the small intestine or the small colon have changed their position in this direction, it may easily happen that the mesenteric veins of the particular loop become compressed to such an extent, that a certain degree of congestion and swelling of the intestinal wall occurs. More active movements of the intestines may now include further loops into the displacement, particularly if neighboring loops cannot get out of the way in consequence of tense filling; this will increase the pressure upon the displaced loops and with it the venous congestion (Wilms).

Predisposing causes also play a rôle in the production of this form of volvulus or torsion, and as such must be mentioned: Greater filling or greater weight of individual loops of intestine (impaction of feces, circumscribed meteorism, tumor, calculi, parasites), relaxed abdominal walls, and adhesions between individual parts of intestines.

**Kinking of the intestines** (turn around the transverse diameter) is rare. It occurs most frequently after loops of intestine have become adherent to neighboring organs or to the abdominal wall. However, the small intestine or the small colon will simply be narrowed without the production of disturbances of circulation and its sequelæ; but the stenosis may finally lead to complete closure as in a case of Avérous in a goat. Kinking of the left divisions of the colon (flexio coli) or to the cecum (flexio cœci) occurs occasionally in the horse without preliminary adhesions; it is then undoubtedly due to the enumerated mechanical factors. It is accompanied by rapid development of congestion, because the vessels of these parts of the intestine are included in the kinking. Kinking of the apex of the cecum, with anemia and necrosis of this part (Johne) has been observed a number of times.

One of the cases of the authors suggests the occurrence of partial kinking in the large colon. The case was that of a horse that succumbed after having suffered for five hours almost continually from colic. Postmortem examination showed kinking of the median wall with its vascular mesocolon, deep into the lumen of the bowel. The kink was found in the region where the transverse divisions go over into the right divisions. The left and the transverse divisions showed a high degree of venous congestion to the point of kinking; nothing abnormal, particularly no thrombi or emboli were found in the arteries of the colon, the anterior mesenteric, or the ileo-cœcolic artery. The origin of this kind of displacement may be explained by assuming that moderate bloating occurred from some cause or other and that the median wall of the dilating division of the colon were pressed inward by some unknown mechanism.



**Predisposition:** The great length of the mesentery of the small intestine in the horse, the fact that most of the colon lies free in the abdominal cavity of this species of animals, the further factor that horses are used for work of various kinds explain the frequency of volvulus and torsion in equines compared with its rarity in other animals. Relaxation of the abdominal walls and more voluminous feed are the reasons why torsion of the large intestine is more common in heavy horses than in lighter breeds under similar conditions.

**Pathogenesis.** The facts as to pathogenesis are on the whole similar to those which prevail in internal strangulation (see page 415). The main difference between internal strangulation on the one hand and volvulus and torsion on the other, lies in the fact that the intestine is not strangulated by another firm tissue in the latter condition, but by other loops of intestine.

**Anatomical Changes.** In torsion of divisions of the colon, one finds the divisions distal from the twisted place distended, dark purplish to blackish red, sometimes spotted with hemorrhages; the mesentery of the two superimposed divisions infiltrated with blood, often gelatinous, the veins in the intestinal wall strongly filled. The latter appears more or less thickened, it is easily torn and is infiltrated with a reddish, serous fluid; the mucosa is blackish red, thrown into folds, pendulous, here and there necrotic. The bowel contains an abundance of hemorrhagic fluid or pasty feces. The congestion is either sharply defined towards the place of constriction or an anemic strip may completely encircle the whole intestine or may be distinct only on two opposite places of the periphery. Exceptionally the congestion may not be very well marked, both on the serosa and the mucosa.

The direction of the torsion may be from right to left, or vice versa, and it may be determined by the relation of the two left divisions of the colon or by the direction of the longitudinal bands. Twists to the right appear to be somewhat more numerous than twists to the left, although in twenty-six cases of torsion Wall saw fifteen to the left and only eleven to the right.

The degree of torsion is very variable. Usually one finds one-half or one entire twist, but several twists are by no means very rare.

The site of torsion (place of twisting, torsion-stricture) is usually found in the transverse colon (diaphragmatic flexure), but, in consequence of falling down of the proximal parts in the region of the right divisions, it may be displaced towards the cecum or to the beginning of the small colon (place of torsion at the root of the colon) and then the cecum may occasionally also be twisted. (Wall, Berlin report 1900-1901.) Longitudinal torsion confined to the pelvic flexure has only been described in

one case (Berlin report 1901-1902); it is, therefore, a great rarity. The place of torsion is also rarely found in the left divisions (fig. 46).

Wall gives the following figures for twenty-six collected cases: Fifteen times place of torsion right at the cecum, seven times in the transverse colon, three times in the left divisions, once at the basis of the cecum, so that the latter had also been twisted; it is possible that the place of torsion is more frequently at the cecum than is usually supposed to be the case, since the finding of the place of torsion in this region is coupled with great difficulties and hence the place may easily be overlooked. In torsion at this place it may appear that only the upper division sinks beside or below the ventral division to become strangulated at its junction with the small colon, without changing its position (authors' observation). In spite of this, venous congestion develops because the vein is compressed either by the prolapsed upper division or in torsion to the left by the tense mesocolon.

**In kinking of the large intestine** the lumen (just like in kinking of any other tubular organ) becomes narrowed or obliterated by the inward projection of the wall, and the parts beyond the kink show signs of intense congestion. In kinking of the apex of the cecum the shut off portion usually becomes anemic and necrotic.

**In torsion of the mesentery of other portions of the intestine,** the mesentery itself is twisted into a cord or there is a volvulus when one portion of the intestines has wound around another portion, and strangulated it like a ring (fig. 47). Parts of intestines strangulated in this manner appear enormously distended and are dark red in color.

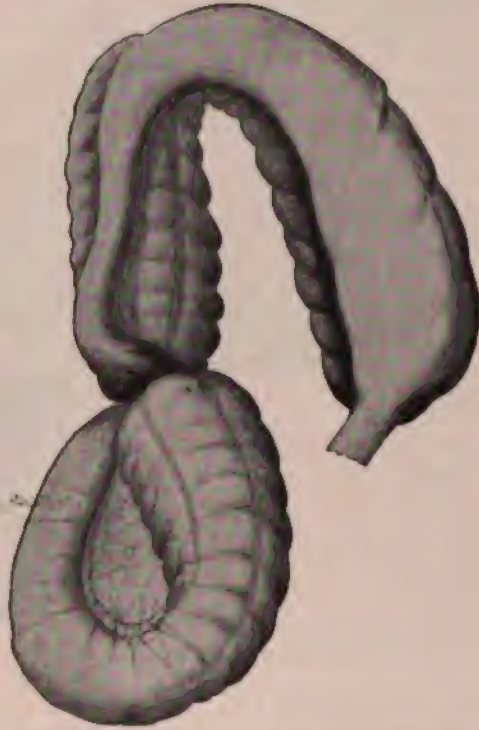


Fig. 46. Torsion of the left divisions of the colon towards the right in a horse also suffering from volvulus of the small intestine. (As a rule torsion occurs more anteriorly.)

The peritoneal cavity contains, in any form of volvulus or torsion, a bloody serous fluid; sometimes circumscribed or diffuse peritonitis is present, occasionally intestinal rupture.

The differential diagnosis of torsion or volvulus must consider hemorrhagic infarction of the intestinal wall in thrombosis or embolism of the mesenteric arteries in those rare cases when on postmortem examination the displacements are not very noticeable or are overlooked entirely. However, a careful examination of the blood vessels, as to the size and position of any thrombi or emboli present, and careful inspection will prevent errors. The sharp demarcation of hemorrhagic infiltration



in a straight or curved line is without significance, because it is likewise seen in thrombotic or embolic infarction (see page 403). It appears doubtful whether one is justified, as Wall claims, to attribute congestion in the colon, in the absence of any torsion, to a stricture which has existed during life, but which has become relieved spontaneously before the animal died.

Postmortem displacement of the intestines can be distinguished from those formed during life, because in the former case signs of congestion are absent.

**Symptoms.** External symptoms of torsion are in general identical in various species of animals with those of internal strangulation of the intestine (see page 417). A decided difference exists in the main in the fact that volvulus and torsion generally lead to a considerable increase of the abdominal cir-



Fig. 47. Volvulus of the small colon. *C*, small colon, *R*, rectum (according to Mollereau).

cumference, because the large intestine or a considerable portion of the small intestine are involved, hence a good deal of bloating will occur.

In the case of **torsion of the colon** in the horse, rectal examination will show a high degree of meteorism. One finds both left divisions, particularly the lower one, considerably distended and reaching up to the upper abdominal wall, so that sometimes it is possible only with the greatest difficulty to force the hand between the bloated bowel and the upper abdominal wall. Firm feces cannot be felt through the much distended and very tense elastic wall. If, however, the patients are examined at once after the onset of the affection or if the latter progresses exceptionally slowly, extensive bloating may be missed for some



time. The other portions of the intestines first remain of normal size, but may often be displaced by the colon out of their proper place (especially the cecum). After a longer duration of the disease the other portions of the intestine likewise become bloated, but never to such an extent as the colon.

The position of the colon either remains normal or one finds the two left divisions displaced towards the median plane of the abdominal cavity, or even towards the base of the cecum. The pelvic flexure is directed either backward or towards one side, or towards the thorax, and the course of the divisions of the colon is either straight or towards one side, or curved in a bow. The mutual relation of the left divisions of the colon remains either normal (in one complete torsion) or their relation has changed so that the smooth upper division, becoming larger towards the thorax, is situated either towards the right or left, or below the much thicker lower division, and presents bands and sacculations. If the pelvic flexure is displaced toward the front, it can be palpated per rectum only in small horses. If, however, the upper division is situated more or less laterally from the lower division, the pelvic flexure is found more or less horizontal on the lower abdominal wall instead of vertical as found under normal conditions. The two free bands of the lower division (one lateral and one median longitudinal band) show the same course as the lower division of the colon itself, but they are also influenced by the position of the pelvic flexure. A spiral course of the longitudinal bands to the right or left can be ascertained particularly in those cases where the spiral formed in twisting reached into the neighborhood of the pelvic flexure, or where the latter has been bent towards the front, otherwise the bands show a normal course. Pulling on the colon or on its longitudinal bands frequently causes pain to the patient. The point of torsion, where the divisions of the colon are twisted into a spiral, tough cord, in a circumscribed place, can only be recognized in those very rare cases where torsion has occurred in the posterior half of the left division of the colon.

According to Jelkmann, in torsion of the colon the hand introduced into the rectum feels the mesentery in the place of the fourth lumbar vertebra as a tense and tender cord which runs from above to the left and downward; one also feels a second tense cord (one of the longitudinal bands of the left lower division) in the region of the left flank. Möller, however, thinks that the characteristic feature is the finding of the two free bands of the bloated left lower division as two tense strings which run in a spiral direction from left to right or from right to left.

**Kinking of the colon** or of the **cecum** may be assumed when the intestinal wall, in some place, forms a sharp angle, and when the bent portion of the intestine is strongly bloated. The place of kinking must, of course, be at a point accessible to rectal exploration, otherwise it cannot be recognized.

Curved bends of the colon, more rarely of the cecum, which are observed in other colicky affection of horses, cannot be looked upon as kinking, because they do not encroach upon the intestinal lumen nor do they produce circulatory disturbances.

**Torsion of the cecum** can probably be often diagnosticated by rectal exploration as was shown in a case which the authors observed.

**Torsion or volvulus of the small colon** (often called the abdominal portion of the rectum) can always be ascertained by rectal examination. If a portion of the small colon, situated very near the rectum, has been closed, the introduction of the hand through the narrowed place is impossible, perhaps a finger may be pressed through. One finds the intestinal wall folded and tender in front of the impediment; bloated loops of intestines can be felt through the wall of the rectum. If torsion has occurred more towards the front, the findings are similar to those in strangulation of the small colon (see page 419), with this difference, that in volvulus with the small intestine the strangulated small colon is surrounded by bloated loops of small intestine.

**Formation of knots and partial mesenteric torsion of the small intestine** may in some cases be ascertained directly by the observation that loops of intestine, which are bloated to the utmost and very tense, suddenly pass over into a folded cord, which, on pressure or on pulling, proves to be tender.

In horses that are not too large, total torsion of the small intestine around the root of the mesentery may be recognized by finding a folded, thick cord, in a vertical plane, supposed to pass through the middle of the left kidney, immediately below the vertebral column, which leads towards the right or left and is connected with a number of very tense loops of small intestine. In some cases one may also find loops of small intestine wound around the caudal portion of the left division of the colon, or around the upper portion of the cecum. In a considerable proportion of cases one cannot find the seat of a torsion or a volvulus of the small intestine, but can only ascertain very great distension and eventually tenderness of some loops, while the rest of the small and the large intestine are normal (circumscribed meteorism). Only rarely does rectal examination furnish a completely negative result, namely, when the displacement has occurred in the anterior parts of the abdominal cavity and when only a short loop is involved.

Rectal examination, or palpation of the abdomen, may reveal conditions similar to those of internal strangulation (see pages 419 and 420) in **cattle, hog and carnivora**. Probst saw the case of a dog with torsion of the rectum and with bloody feces (probably the torsion was originally not complete in this case). The authors saw a similar case in a horse with torsion of the rectum.

**Course.** The clinical picture sometimes develops during the course of other colicky affections, but, as a rule, it has a very sudden onset. As in displacements of the intestines in

general, either intense symptoms of restlessness occur at once or they increase gradually in intensity. The disease is of short duration, since death occurs rapidly, particularly in displacements of the small intestine, sometimes after a few hours, usually towards the end of the first or during the next day. With torsion of the large intestines, particularly with torsion of the small colon, horses may live two or three days. In other domestic animals the course is similar to that in strangulation of the intestines.

The possibility of spontaneous recovery cannot be excluded *a priori*; but spontaneous reduction may occur only in displacements of smaller sections of the intestines and only in the beginning of the disease, that is, as long as bloating in the affected portion does not yet exist, which would, of course, make a spontaneous recovery impossible.

**Diagnosis.** Torsion and volvulus can only be diagnosed beyond doubt if rectal examination in large animals, or abdominal palpation in smaller ones, reveals the place of twisting or entanglement. The other signs, such as a spiral course of the longitudinal bands of the colon, changes in the interrelations of the left divisions of the colon, and their displacement into other portions of the abdominal cavity has no great diagnostic value, since this is also found in other colicky affections. If, however, the left upper division of the colon of a horse is found along its whole palpable length, obliquely below and to the left of the lower division, then the existence of torsion of the colon is very probable.

As already mentioned (see page 433), Jelkmann believes that the presence of a tense, tender string running obliquely from above downward and to the left in the plane of the fourth dorsal vertebra, and of another tense string in the region of the left flank, is very characteristic. Möller considers the spiral course of the longitudinal bands as decisive. Forssell, on the other hand, thinks that the relation of the left divisions of the colon and the course of the band is the determining diagnostic factor. It must, however, be pointed out that the longitudinal bands of the left lower division may show the same course in every case of bloating or in excessive filling of the colon, because the much-dilated lower divisions will rise up to the left kidney, the upper division is, at the same time, displaced to the right or to the left, and in consequence of an increase in length, these divisions, and also the pelvic flexure, become displaced to the right or left and towards the thorax, which compels the stretched longitudinal bands to assume a spiral course without, however, encroaching upon the intestinal lumen or without the production of a venous congestion. This is proven by the observations of the authors and by those of other authors (Larsen, Klett, Behrens), who also believe that torsion of the colon can, as a rule, not be diagnosed with certainty by a rectal examination alone.

The direction of the torsion is determined by Möller from the course of the longitudinal bands. In torsion to the right, the band of the lower divisions lead from in front towards the back and to the right; in torsion to the left in the opposite direction. Forssell gives the following rules;



1. If the left upper division lies to the right of the lower division and if it passes here obliquely forward, upward and to the left, then we are dealing with a half twist towards the right. If the left upper division passes obliquely forward and downward and to the left, then we have a twist to the left of more than  $90^{\circ}$ .

2. If one feels the left upper division to the left of the lower division and if it leads obliquely forward and downward and to the right, then we are dealing with torsion to the right of more than  $90^{\circ}$ ; if it leads obliquely forward and upward and to the right, then we are dealing with a half torsion to the left.

3. In those rare cases where the left lower division completely covers the upper division, the spiral course of the band and indentations of the left lower division which may be present are the deciding factors.

Concerning the details of diagnosis, including differential diagnosis, the reader is referred to the subject of internal strangulation (see page 422). Even where rectal examination or external abdominal palpation give information, the affection cannot always be differentiated from internal strangulation, but this is of no great importance, since the therapeutic procedures are the same in both cases.

**Treatment.** The same principles as those laid down for the treatment of internal strangulation (see page 423) govern on the whole the treatment of torsion or volvulus. However, the very nature of these affections brings it about that, except in a case of torsion of the colon, reposition from the rectum is even less frequently possible than in internal strangulation.

In torsion of the colon one should always roll the horses in the direction of the torsion whether it be diagnosed positively or only suspected. Forssell has recently had some good results with this procedure.

**Relief of torsion of the colon by rolling,** according to Forssell, is brought about as follows: According to the Stuttgart method the horse is best placed on the side corresponding to the twist of torsion, that is, if torsion is towards the right, the horse is placed on the right side. Before this is done the bloated colon must have been punctured either through the left abdominal wall or through the rectum (see page 363). Then the arm of the operator is introduced into the rectum; he next tries to fix the pelvic flexure with his fingers (in mares one may make an incision into the vagina for this purpose); indeed, according to Forssell, the intestine becomes fixed by its own weight. Then the horse is rolled on its back and abdomen till the intestine gets into its proper position. (A horse had to be rolled nine times in a case of Forssell before replacement occurred.) To prevent rupture of the rectum, the fixation with the fingers must be interrupted from time to time. The reposition of the bowel is usually announced by the expulsion of intestinal gases, although sometimes flatus does not immediately occur. The restlessness persists for a few hours if the rolling was performed a few hours after the onset of the disease; if, however, the horses have been sick for fifteen or twenty hours, they become quiet almost immediately after reposition. The after treatment consists in starvation or in very careful feeding for several days.

The therapeutic value of rolling in torsion of the colon cannot be judged accurately on account of the uncertainty of the clinical diagnosis and the comparatively small number of observations in this respect. Forssell reports seventeen recoveries in eighteen cases so treated. Other authors (Behrens, Hummerich) have, however, not been able to get such brilliant results. Hummerich and Kalcher, on the other hand (see pages 363 and 372) have been successful in treating many

cases of primary intestinal meteorism and impaction by rolling which indeed is nothing more or less than a particularly energetic abdominal massage; these diseases may, as pointed out, easily be confounded with torsion of the colon on account of the frequent change in the inter-relations of the left divisions of the colon. It appears at least questionable, therefore, whether indeed the majority of cases of torsion of the colon can be cured by rolling. If the place of torsion is in the transverse colon or more towards the periphery in the left divisions, then reposition by rolling may easily succeed; in torsions of the right division, which appear to be more common, rolling cannot have any immediate effect, because the place of torsion does not coincide with the place around which the colon turns in rolling. Only very accidentally might the normal position be then restored. (In one of the authors' cases with a torsion of all divisions of the colon to the left, repeated rolling after twice puncturing the bowel remained without any effect at all.)

In consideration of experiences with the correction of torsion of the uterus, it appears rather strange that rolling of horses with a supposed torsion of the colon, in the wrong direction, should not be at all detrimental; that it should not be capable of producing torsion of the colon in horses with other intestinal affections, and that it should favorably influence bloating of the cecum, which is claimed to be caused always (?) by torsion of the cecum around its long axis.

The correction through the rectum of torsion of the colon, according to Jelkmann and Möller, is performed on the standing animal after a preliminary puncture of the intestine. The posterior abdominal portion of the animal is first elevated, the hand is then introduced into the rectum, and guided towards the left abdominal wall. The hand then tries to push forward and inward the accessible loops of the small colon and those divisions of the colon which are above the former; if the median line has never been reached the hand is guided upward, whereupon, in favorable cases, the colon glides into the place of the loops of the small colon which have been pushed aside; in this manner the torsion may become relieved.

This procedure was recommended by Jelkmann and is indicated only in torsion towards the left. Möller proceeds in another manner. He pushes with the hand, introduced into the rectum, the upper division, which is twisted towards the left, upward and at the same time the lower division downward and to the left.

The experiences of Larsen, Keutzer, Forssell, Straube, however, and the observation of the authors show that, even with a good deal of force and persistency, torsion cannot be corrected because the colon is such a heavy body that it cannot easily be moved in the closed abdominal cavity where neighboring loops of intestines at once occupy every space that might be made free. Forssell also calls attention to the ever present danger of rupture of the rectum. Jelkmann, Möller, Malkmus, Zippel and others have had good results in those cases which belonged to the type of cases recommended for their procedures by Jelkmann and Möller.

Kinking of the pelvic flexure can be corrected from the rectum by pulling towards the pelvis (Diem). Sigl has relieved a case of torsion of the small colon by the injection of water into the section situated in front of the impediment.

When a reliable diagnosis has been made laparotomy is usually the only indicated procedure except in cases of torsion and kinking of the large intestine. Aside from its danger to life, however, even this procedure cannot always produce the desired result, as is evident from the nature of the disease itself. The experiences of the authors show that volvulus or extensive torsion of the mesentery cannot be cured even by this operative procedure. In torsion of the colon, laparotomy with subsequent rolling may be indicated (Forssell). Concerning the indications for laparotomy the reader is referred to the subject of intestinal strangulation (see page 425).

In a case in a horse where loops of the small intestines had become twisted around the small colon Meschkow was able to replace the displaced loops after opening up the right flank; recovery took place. Sigl had a similarly good result in volvulus of the small colon. Andebert replaced divisions of the colon, alleged to have been displaced, with the hand introduced through an incision into the wall of the vagina. Plósz & Marek were unable in two cases to reduce a mesenteric torsion of almost all of the small intestine after laparotomy. Ries performed laparotomy



in cattle in two cases; he succeeded once in correcting a displacement. Jöhnk succeeded in this manner to correct invagination and torsion which were present simultaneously.

Since reposition of twisted divisions of the colon might be brought about spontaneously by rolling, moderate rolling of the patients may be permitted, but in torsion or volvulus of the small intestine or of the colon this would only cause displacement of the remainder of the intestine. In these cases, and in torsion or volvulus in general, the pain should be relieved by narcotics (morphine, chloral hydrate). Intestinal puncture (see page 363) is indicated, without any reference to further treatment, whenever there is intense bloating; some cases of intestinal flexure (see page 429) or of other displacements of the large intestine may occasionally assume their normal posture after the escape of gases. The use of laxatives is contraindicated. The favorable results claimed by Oeller and Ohler can not be judged objectively on account of the insufficient clinical diagnosis in these cases.

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## 26. Intestinal Invagination. Intussusception. Invaginatio intestini.

By intussusception one understands a stenosis or occlusion of the intestinal lumen which occurs suddenly and is caused by the invagination of one section of intestine into a neighboring part, followed by a venous congestion in the invaginated portion.

**Occurrence.** Invagination is found most frequently (but even then as a rare occurrence) in cattle and somewhat more rarely in dogs, very rarely in the horse and in the hog. Sometimes it occurs in fowl (Kitt, Johns, Klee).

In the Prussian army during 1892-1908, invagination was seen in ninety-nine cases among 71,532 cases of colic (0.14%); in the Berlin Clinic, 1897-1907, there were nine cases of invagination among 8,686 cases of colic (0.1%); in the Dresden Clinic in 3,336 cases of colic, five (0.15%); and in the Budapest Clinic invagination was represented with 0.1%.

**Etiology.** If for some reason or other a piece of intestine contracts more strongly and remains in this condition for some



time, it may slip into the section situated immediately behind it. In consequence of the contraction of the circular muscle fibers, in peristalsis progressing towards the anus, a piece of intestine becomes thinner and longer, the next posterior piece, however, becomes wider and shorter in consequence of contractions of the longitudinal muscle fibers; the thinner and longer section can easily slip into the wider and shorter piece, if this becomes more or less fixed, or less motile, or if the section nearer to the stomach meets with an impediment at its proximal end. Nothnagel has shown by animal experiments that such invaginations may occur in normal peristalsis, that they are, however, at once replaced, because only short portions are invaginated and the contractions do not last long. Permanent invagination occurs only in abnormally active and energetic peristalsis. Hence all stimuli which increase peristalsis may bring about invagination indirectly.

The peristalsis may be increased in all or in certain parts of the intestines, and become convulsive in sections, after a cold, after the ingestion of ice cold water or cold feed (frosted grass, frozen bulbs), in the course of an intestinal catarrh or enteritis, in the presence of intestinal parasites, foreign bodies, tumors of the intestinal wall, and in attacks of intestinal colic. These conditions, in combination with predisposing causes to be mentioned presently, may bring about invagination. (Wagenheuser has seen seven cases occurring at the same time in foals after the ingestion of frosted grass.)

In agonal states the increased amount of carbon-dioxide of the blood which causes increased peristalsis not infrequently produces invagination; this, however, occurs so late that no more circulatory disturbances can develop in the invaginated portion of the intestine (agonal invagination).

It is not impossible that in certain cases a section of intestines, which is just contracting, may become invaginated into a neighboring piece by a sudden great increase of intraabdominal pressure. This may explain those cases in which horses suddenly fall sick in jumping, and cattle in running about, or during parturition, or in drawing a load uphill. Even in these cases, however, the peristalsis which has become more intense, is probably the immediate causative factor.

Both veterinary and medical authors still claim frequently that invagination is brought about on certain occasions in such a manner that sections of intestines situated towards the anus become paralyzed and that sections situated toward the stomach, which are quite motile, slip into the paralyzed portion. In this manner sections are paralyzed during the agonal stage and other sections are still very motile. The authors cannot indorse this so-called paralytic theory. If it were true then invagination should be equally frequent in all species of animals, and young animals, in whom even insignificant stimuli are liable to excite lively peristalsis, should not be affected preferably. One would not expect the great rarity of the affection in horses which offer so many chances to partial intestinal paralysis (thrombosis of the mesenteric vessels). It would also not be obvious why very long sections of the intestines are frequently invaginated. Nothnagel could not verify the paralytic theory in his experiments.



As predisposing causes must be considered the firmer attachment by a short mesentery of some portions of the intestine, a heavy tumor, adhesions, occasionally also bloating.

The seat of an invagination is most frequently the small intestine, since it is especially subject to stronger contractions; more rarely the ileum becomes invaginated into the cecum, exceptionally into the colon (Johne); in horses, cattle and dogs, the apex of the cecum into its body; in horses and exceptionally also in dogs the cecum into the colon, rarely the small colon into itself or into the rectum. There has also been observed invagination of the duodenum into the stomach (H. Bouley in a horse, and Peuch and Cadéac each in one dog). This occurrence must have been due to antiperistalsis which may also cause invagination in other portions of the intestinal tract into a section situated towards the stomach.

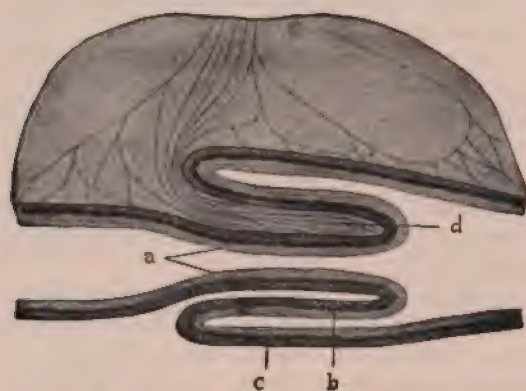


Fig. 48. Diagrammatic representation of invagination. *a.* internal, *b.* external portion of the intussusceptum, *d.* its mesentery, *c.* intussusciens.

### Pathogenesis.

Wherever invagination occurs, the intussuscepting portion will, of necessity, draw along its mesentery with the blood vessels of the latter (Fig. 48). The intussuscepting portion and its mesentery will be subjected to a certain amount of pressure, depending upon the caliber of the intussuscepted section; this will cause congestion,

swelling and subsequently a hemorrhagic serous infiltration of the drawn-in portion of the intestine and its mesentery. The extravasating hemorrhagic fluid gets into the lumen, also between the invaginating and the invaginated portions, and also to a small extent into the free peritoneal cavity at the place of entrance of the invaginating portion.

Passive congestion, which increases rapidly in the intussuscepted portion, causes marked convulsive contractions with colicky pains, and the former may cause an increase of the portion invaginated towards the anus. This is favored by the fact that while the circular fibers of the intussuscepted portion contract, the longitudinal fibers of the intussuscepting section likewise contract. If the place of invagination is somewhat fixed, the latter section will slip over the former. Contraction of the circular fibers of the invaginating portion of the intestine may cause an increase in the invaginated portion, because the con-

tracted elongated bowel may, at its anterior end, slip further over the invaginated bowel. These factors may sometimes bring it about that the invaginated intestines may be several meters long. The increasing contractions of the invaginated intestine with its mesentery causes continuous pain which, as usually in displacements, becomes augmented by external or by increased intraabdominal pressure. However, the pain is, as a rule, not very intense.

The lumen of the invaginated bowel is considerably diminished from the start, and it may not rarely become entirely obstructed in consequence of swelling of the intussuscepted portion, together with its mesentery. The larger the intussuscepted bowel, the less the lumen of the intussuscepting portion will be diminished. Diminution or obstruction of the intestinal lumen excites more active, later on convulsive contractions in the intestines situated towards the stomach; these contractions are moderately painful. The peristalsis of the sections situated towards the anus only ceases when the invaginated portion has lost its contractility; this occurs in consequence of intense infiltration, necrosis, secondary peritonitis, or excessive compression; usually, however, only after a number of hours and even days. As long as the invaginated bowel moves, the peristalsis progresses towards the anus, even if complete obliteration supervenes eventually, because the strangulation is not so severe that the peristalsis may not progress from the invaginating toward the invaginated portion. These contractions may be sluggish; they continue for some time or even until the end, and propel the serous, hemorrhagic fluid, that is extravasated into the lumen, towards the rectum.



Fig. 49. Invagination in a horse.

The nutritive disturbances which are caused by the congestion of the intestinal walls may be very variable in degree, according to the nature of the affection, and lead to similar consequences as they are found in internal strangulation; the im-



mediately affected portions of the peritoneum are, however, less endangered (see page 416).

In those very rare cases in which the patients remain alive for a long time, the invaginated necrotic portion of intestine may occasionally become detached; if this does not occur and if there has not been complete stenosis, the latter will then occur.

**Anatomical Changes.** The two invaginated portions of intestines form a curved, screwlike, or tortuous cylinder (Figs. 49 and 50) which is firm, sausagelike in consistency or fluctuating. It is usually several centimeters long and occasionally measures several meters long. In the latter case one can feel a sausagelike body in the affected part. The bowel looks normal externally, or purplish to dark bluish-red, because the folding of the mesentery also compresses the veins of the outer portion. At the end of the cylinder one sees the place of entrance of the

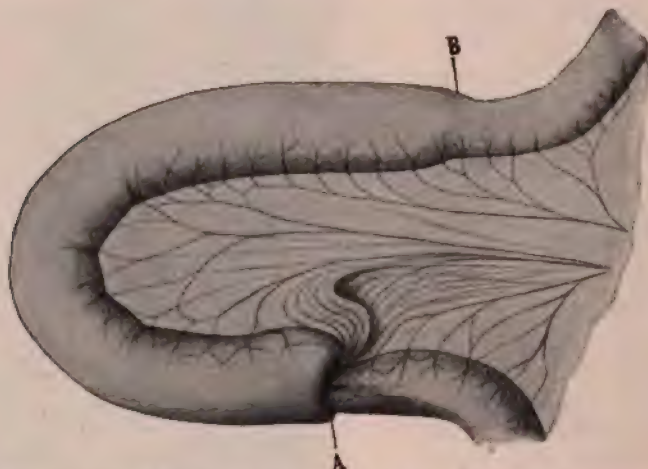


Fig. 50. Invagination of the small intestine in a dog. A, Place of entrance, B, conical end of the invaginated bowel.

narrowed portion of bowel with its mesentery (intussusceptum) into the most external layer of the wall composed of three layers (intussusciens, see Fig. 48). Wall and mesentery of the intussusceptum exhibit intense venous congestion. Where the ingoing and outcoming portions of the intussusceptum are in contact with each other, fibrinous peritonitis is soon developed, hence the pieces of intestines can be drawn apart only with difficulty or not at all. An invagination which has been formed during the agonal stage can easily be reduced and does not show congestive or inflammatory changes. Invagination is, as a rule, single, but it may be double, triple or quadruple. Sometimes invaginations are found in several places. Finally, one may find the evidences of a general peritonitis.

**Symptoms.** In horses invagination produces on the whole the same clinical picture as internal strangulation (see page 417). In invagination of the cecum into itself or into the colon defecation is not entirely suppressed, or at least not continuously; the animal may even survive; but after the violent symptoms have disappeared those of intestinal stenosis remain behind (see page 389).

Rectal examination often shows important differences between invagination and other intestinal displacements. Circumscribed meteorism is absent until the end, except in protracted cases, when all the intestines become moderately bloated. In invagination of the small intestine into itself, the hand introduced into the rectum feels a thick, elastic-firm, painful, sausage-like body which may be wound up like a snail, of the consistency of meat; at other times a more or less fluctuating loop of intestine in which, on firmer pressure, a sausagelike body may be felt. Sometimes only the place of entrance can be reached, where the intestine becomes thinned out to a cord and disappears in a thick fleshy ring. In invagination of the ileum into the cecum, one feels in the head of the cecum a body as large as a wrist, somewhat tender, movable and of tough-elastic consistency (Klett). Occasionally invagination of the cecum into itself may be felt, or of the cecum into the colon.

In those rare cases where invagination did not occur towards the front and where peristalsis did not cease in the portions towards the anus soft hemorrhagic feces are seen.

In **cattle** the clinical picture is also quite similar to that found in internal strangulation (see page 419). Kolb noticed in one case stretching in urinating in the manner seen in male horses. The feces are at first normal, later on they become thinner and occasionally hemorrhagic; in the further course defecation ceases, or glairy or bloody mucus and fibrinous masses are voided under great efforts. Rectal examination reveals conditions similar to those found in the horse.

In **dogs** the clinical picture of invagination differs from that of internal strangulation only by difficult defecation and bloody fetid feces are observed. The general condition, pulse and respiration of the animals are little or not at all disturbed for one or two days; the appetite is, however, suppressed from the start. Parent saw prolapse of the invaginated bowel at each defecation in a young dog with invagination of the colon into itself. Palpation of the abdomen reveals an elastic-firm, cylindrical, sometimes curved, painful body which can be moved freely in every direction. One can sometimes feel the conical end of the invaginated part and on the other side the turning point of the external tube of the sausagelike body (Fig. 50).

In a case of Hutyrá's the emaciated animal showed for a long time obstinate bloating, the appetite was diminished, ingested soup or milk was not expelled by vomiting, daily defecation produced thin-fluid stools. The postmortem examination showed a long-standing invagination of the colon.



**Course.** After the disappearance of the abdominal pains, which are at first very intense, several hours elapse (in cattle usually six to twelve hours), occasionally several days (particularly in horses), then the animals become quiet, and they die from complications which have developed. In horses several attacks of colic may be observed exceptionally, occurring at brief intervals, while some authors claim that in cattle the disease may run its course without any pain. Pains may, however, be overlooked of course in animals which are not always under observation (at night).

The disease lasts usually six to nine days in cattle and may rarely be prolonged to two weeks, while a short course of only a few days is rare. In horses invagination of the cecum into itself or into the colon may run a course of six weeks (Panthe) or even of months (Colin); during these long periods the animals present the symptoms of intestinal stenosis.

Recovery is rare, but possible if the invaginated portion becomes cut off at its oral end and is voided with the feces, in cases where adhesions have formed between the serous layers at the entrance of the invagination. Cases of this kind have been reported in horses (Verrier, Rackow, Martin, Hochstein, Perkuhn) and also in cattle. Stenosis of the intestine usually follows this course.

**Diagnosis.** Intestinal invagination may be diagnosticated with certainty by positive findings on rectal examination; in very rare cases a diagnosis may be made later in the course of the disease, on account of the expulsion of pieces of intestines with the feces. Hemorrhagic feces with mucus or even masses of fibrin, also symptoms of intestinal obstruction and signs of general disease can, in spite of an early disappearance of the restlessness, create only a suspicion of invagination; they cannot be relied upon for a definite diagnosis.

On the basis of the results of rectal examination, the affection may be confounded with impaction in the small intestines and also, in cattle, with impaction in the large intestine; impacted intestinal loops feel more doughy or harder, less elastic, nodular, not painful, and often several loops show this condition. One also finds occasionally thick-walled, painful elastic portions of the intestines in membranous enteritis, but the symptoms of intestinal occlusion are then missing. Invagination in cattle cannot always be differentiated from volvulus or from obstruction of the intestines with coagulated blood; this is, however, immaterial as far as further therapeutic procedures are concerned. In dogs the affection might be confounded, after abdominal palpation, with impaction in the large intestine; but in obstipation we find a soft-firm or then a hard, nodular, cylindrical mass, not movable, not elastic and, as a rule, not painful; defecation does not occur. The affection of invagination in dogs may more easily be confounded with obstruction of the



intestines by foreign bodies, so that an exploratory laparotomy alone can decide the diagnosis. In cows the differential diagnosis must exclude torsion of the uterus (Albrecht).

**Treatment.** The only treatment that promises any success at all is laparotomy. After opening the abdominal cavity (in cattle in the right flank, in dogs and possibly also in foals, in the median line, in horses at a point next to the seat of the invagination) the invaginated intestine is located and drawn out. The loops are then reduced or, if adhesions have formed or there are signs of necrosis, the affected portion of the intestine is resected and the ends of healthy intestine are united by a suture. In separating an invaginated portion it is advisable not merely to pull, but to press at the other end.

The operation was first performed in cattle by Luscan in France and by Meyer in Germany, and good results were obtained in this animal. (Luscan, Meyer, Taccoen, Hafner, Matthieu, Perdan, Guittard, Schmidt, Schiel, Jöhnk and others); Michener cured invagination in young foal; Plósz obtained good results in several dogs by laparotomy made in the median line.

Whether it be possible to free the invaginated intestine with the hand in the rectum in larger animals, or through the intact abdominal wall in smaller animals, is a question which cannot yet be answered.

The use of laxatives is contraindicated; they can only do harm. The injection of water might be tried, but one can expect good results only in certain invaginations of the small colon or of the rectum.

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## 27. Colicky Affections in Horses.

The term colic means intestinal pain, according to its etymologic derivation (*dolor coli*, *colica*, *neuralgia colica*, *enteralgia*, *enterodynia*). By typical colicky pains one has always meant in human medicine peculiar, cutting, pinching or tearing intestinal pains (*Bauchzwicken* [German]) which come on suddenly in the form of an attack and disappear again suddenly after a certain period of time. Nothnagel has shown and the authors have become convinced from the results of their experiments and rectal examinations that genuine colicky pains are always due to convulsive (tetanic) contractions which cause compression of terminal sensory nerves. The experience of veterinary practitioners leaves no doubt that contractions of the muscularis of

be more convenient to treat a patient by routine for colic without an exhaustive tedious examination and without taking into consideration the real requirements of the case, but this procedure will surely do harm in many cases, where treatment would be successful if it were administered as indicated by the particular underlying affection.

One should, therefore, only speak of "colicky affections," "coliclike affections" of the stomach, intestines, gall ducts, ureters, for the sake of brevity and for descriptive purposes, in the case of diseases which may have either true colicky pains or coliclike pains as one of their symptoms. Otherwise the affections should be treated separately and this plan has been carried out in the preceding chapters. Cadéac has considered these affections in the same spirit.

After the necessity of separating different forms of colic had become obvious, a number of sub-groups were formed. Diseases of the stomach and intestines which are characterized by abdominal pains and usually also by disturbances of defecation or by suppression of peristalsis have been distinguished as genuine colic, from false and from symptomatic colic. With the name false colic were designated painful affections of the peritoneum, the spleen, the kidneys, the internal organs of generation, with the name symptomatic colic attacks of pains occurring in the course of such diseases as anthrax, purpura hemorrhagica, poisoning, gastro-intestinal inflammations. If the true meaning of the term colic is considered, one will fully understand the impropriety of such classifications.

If, in closing the discussions of diseases of the stomach and intestines, which may be the cause of symptoms of colic, the authors here add a special chapter devoted to a general description of colicky affections, they do so from various motives. Without such a chapter they could, in the first place, not clearly define what is meant by genuine colicky pains and differentiate them from other pains which are not of a true colicky nature, but which manifest themselves in a similar manner. Besides unnecessary repetitions in describing the symptomatology of diseases of the stomach and intestines may be avoided by treating of symptoms of colic in a separate chapter. And it is finally not at all superfluous to treat collectively the differential features of all diseases which may give rise to the symptoms of colic.

Diseases of horses leading to the symptoms of colic are very frequent. This is shown among other observations by the statistics of the Prussian army, showing that three to five per cent of all horses annually become affected with "colic," while the proportion of the total morbidity to "colic" is like 100 to 11-14. In veterinary clinics one-half or even more of all horses brought to the clinic suffer from colic; this very high figure is due to the fact that horses suffering from other diseases which lead to less obvious symptoms than "colic" are not brought to the clinic so frequently.

The mortality of colicky affections varies within wide limits because the underlying diseases are different under different conditions and in various localities. This figure varies between 10 to 17 per cent or perhaps even a little higher; it is, on an average, about 12 per cent.

Various circumstances afford the reasons why colicky affections are so common in horses. Horses appear to be more sen-

sitive to pain than other species of animals. It is also part of the nature of the horse to react to disagreeable or painful sensations in a very marked manner. Moderate pains often do not become noticeable at all in other species of animals, although they are felt.

Another cause of the frequency of colicky affections in the horse are the anatomical conditions of the abdominal organs. The stomach is comparatively small, the pylorus is situated between the blind pouch of the stomach and the stomachlike dilatation of the large colon; this predisposes to overloading or dilatation of the stomach. The unusual length of the mesentery of the small intestine and the mostly free arrangement of the voluminous large intestine of the horse, predispose to certain displacements of the intestine under certain conditions (strangulation, volvulus or torsion).

The physiologic condition of nutrition in horses likewise contributes materially to the occurrence of colicky affections. As shown by the experiments of Ellenberger, Tangl and Scheunert, part of the gastric contents pass into the intestines very early, even towards the end of a meal; the food is chewed only once and not particularly well prepared, it may consequently easily exert a deleterious influence upon the intestines, either by undergoing an abnormal fermentation and irritating the intestinal mucosa or by easily becoming inspissated. Part of the ingested water also enters the small intestine immediately.

Very important are the methods of using horses and the amount and character of attendance they receive. No other domestic animal is so much exposed to errors in diet as the horse, especially in big cities where horses often receive spoiled food or improper food substitutes; often their meal time is not observed regularly; livery stable and dray horses must often work immediately after having been fed. The use of the horse as a work animal also brings about its frequent exposure to the inclemencies of the weather and to cold.

An important rôle must finally be attributed to thrombosis of the mesenteric arteries which occurs practically without exception only in the horse, but is very frequent in this animal. This affection accounts for a considerable percentage of the "colicky" diseases.

**Symptoms of Colic.** Horses frequently look around towards the posterior abdomen; they trip about restlessly, groan, paw and stamp with their front legs, and kick against the abdomen with their hind legs; if they become quiet for a time, they stand with the back arched, the neck and head stretched; they place the feet under the abdomen, switch the tail and are dull and apathetic. If the pains come on during motion, the animals have a short stiff gait; they do not like to move, soon stand still and can be made to move onward only by energetic urging.



During the periods of restlessness the animals try to lie down, doing so with great care; they may rest upon the abdomen with their legs drawn under it, or they rest upon the back or roll from side to side. In other cases patients suddenly and recklessly throw themselves down upon the ground, kick with the feet and roll very recklessly. Often the animals assume abnormal positions; they kneel on their front legs or sit on their haunches; they stretch out their front legs and elevate the thorax and head (sitting dog-fashion) or they stretch out their front legs and rest on the sternum.

The symptoms usually come on in the form of paroxysms, between which the animals are quiet and worn out; frequently, however, they recover sufficiently and begin to eat, but a renewed attack soon interrupts the meal. Sometimes the restlessness is remarkably violent, the patients continually change their positions with hardly noticeable pauses; they constantly throw themselves on the floor and assume various positions; jump up again, kick, paw, rear and bite into various objects within their reach. During such violent attacks one may observe grating of the teeth, trembling, nervous movements (pressing forward, circulatory motions, shaking of the head).

Symptoms on the part of various organs depend upon the causative morbid conditions. These symptoms have been described in the preceding chapters.

**Differential Diagnosis.** Since some of these symptoms, eventually also all of them, may be due to genuine colic or to various other disagreeable sensations, it is always necessary, whenever symptoms of colic are present, to examine methodically all of the organs which may be the seat of the underlying affection, and to arrive at a differential diagnosis according to the principles laid down in the preceding chapters. Sometimes it may be necessary to ameliorate or remove a dangerous symptom even before the institution of the general examination.

Symptoms of restlessness may be caused by the following morbid conditions:

1. Diseases of the stomach: Acute and chronic gastric dilatation (overloading of the stomach, including stenosis of the pylorus), gastritis, gastric ulcer, parasites in the stomach.
2. Intestinal diseases: Acute enteritis (rheumatic intestinal spasm, spasmodic colic), occasionally chronic intestinal catarrh, enteritis (including specific acute infectious diseases and inflammations caused by poisons), meteorism, impaction, internal obstruction, stenosis, thrombosis of mesenteric vessels, strangulation (also incarcerated abdominal hernia), torsion and volvulus, invagination, intestinal worms.
3. Acute peritonitis.
4. Diseases of the liver and bile ducts: Lodgment of gallstones, acute, occasionally also chronic hepatitis, rapid increase

in the size of the liver (for instance in consequence of hemorrhage), rupture of the liver.

5. Diseases of the urinary organs: Acute nephritis, nephritic abscess, pyelitis, obstruction of the ureter, inflammation of the bladder, occlusion of the urethra.

6. Diseases of the sexual organs: Torsion of the uterus, spasm of the uterus caused by movements of the fetus, labor pains in normal delivery and in abortion.

7. Diseases of other organs: Pleuritis (very rare!) certain diseases of the esophagus (spasm, obstruction, stenosis, dilatation), irritation of the rectum and its neighborhood by worms (oxyuris, gastrophilus larvæ), paralytic hemoglobinemias; also long-continued hunger and great exhaustion from work.

In making a diagnosis, symptoms of colic are only of minor importance, because they do not permit the determination of the underlying individual affection, or even of a group of allied affections. Almost continuous restlessness is, however, observed most frequently in acute gastric dilatation, in displacements of the intestines, in the grave forms of thrombosis of intestinal vessels and in certain forms of obturation of the intestines. Unnatural positions are seen, especially in forward dislocation of the diaphragm, in intense intestinal tension or pain in the abdomen (displacements of the intestines, impaction, intestinal calculi).

Icteric discoloration of the mucosa with simultaneous restlessness points to disease of the duodenum, of the bile ducts or of the liver.

Elevation of temperature from the onset points to an inflammatory or infectious character of the disease, while a subsequent rise of temperature may be caused by a secondary inflammatory process.

In cases which have not yet progressed too far, a nearly normal frequency and character of the pulse permits the exclusion of grave inflammatory and infectious processes, also pressure upon the diaphragm, and usually also displacements of the intestines and the grave types of thrombosis. In the further course of the disease and in young animals these features can, however, not be utilized for a differential diagnosis. The distribution of surface temperature usually runs parallel with the character of the pulse and with the condition of the respiration. The examination of the thorax occasionally furnishes data for a diagnosis of diaphragmatic hernia.

Belching, retching, vomiting and a sour smell of the expired air speak in favor of primary or secondary affections of the stomach.

Enlargement of the abnormal circumference may be absent in primary or secondary meteorism or it may be hardly observable if the abdominal walls are very tense or if the meteorism is confined to a small portion of the intestine; marked

extension of the abdomen is usually caused only by bloating of the large intestine.

Percussion of the abdomen hardly assists in arriving at a diagnosis.

The intestinal sounds are of greater prognostic than diagnostic value. The absence of peristalsis in certain portions of the intestines cannot be determined from the behavior of the intestinal sounds, because, unless quite feeble, the latter are conducted from one spot to all parts of the abdominal wall. If, however, the sounds are persistently more frequent, and loudest over certain sections of the abdomen, one may conclude that the intestines of this region are in a condition of energetic peristalsis.

Complete constipation, coming on simultaneously with or soon after restlessness, speaks for obstruction or paralysis of the intestine; the absence of these symptoms at the beginning does not, however, exclude these affections. Retarded defecation, and even complete constipation, is observed also during the later course of disease of the stomach. Marked straining at defecation is observed most commonly in diseases of the rectum and in peritonitis. The character of the feces assists in some cases in arriving at a diagnosis.

Rectal examination furnishes the most valuable data and it should never be neglected. There is not a single reason in favor of its neglect and many cases can be diagnosticated correctly on the basis of a proper rectal examination.

To make a careful rectal examination one should first inject two or three quarts of lukewarm water into the rectum; this relaxes the rectal wall and makes its internal surface slippery. Before introducing the hand the neighborhood of the anus is inspected and tumors, parasites or blood are noted if present. The oiled or greased hand is introduced by overcoming the resistance of the rectum with the fingers closed into a cone. It is then advanced carefully until the arm has been pushed in as far as the elbow; while this is being done one can ascertain, by palpating the rectal wall, whether the latter has been torn. The hand then progresses in the direction of the thorax to find out whether the rectum is empty (obstruction). Then palpation is made through the rectal wall in order to examine the urinary bladder, the two abdominal rings (in mares the ovaries), the accessible portions of the small and large intestines, the posterior or mesenteric root, the posterior, upper portion of the spleen, the left kidney and, in horses that are not too large, the anterior mesenteric root and the vessels contained in it. A man of medium size is able, in not too large horses, after introducing the arm up to the shoulder, to palpate the anterior portion of the left kidney and from there downward and laterally a spherical segment of the abdominal cavity. If possible the examination is made on the standing horse. Very restless horses must be first quieted by an injection of morphine or by chloral hydrate, or they must be restrained.

**Treatment.** In order to avoid serious errors, treatment must always be directed against the underlying disease, which has either been determined with certainty or at least with good probability. The variable nature of the causes of colicky pain excludes any uniform method of treatment; it is, however, always advisable to take the patients to a roomy closed place well littered with dry straw, to prevent reckless throwing down and impetuous rolling partly by narcotic means, partly by proper



From the oblong ova (Fig. 51), opening by a lid and glued to the hairs in a downward direction, there issue, after a few days, slender larvæ composed of thirteen segments. The horses lick these larvæ, probably because of itching, get them into their mouths and ingest them with the food and water into the stomach; here the larvæ bore into the mucosa and develop. According to another view, the larvæ wander in the mouth from various portions of the body of the horse, or to a place where they can be licked off. After about ten months, that is, between May and September, especially, however, in July, they become detached from the mucosa and get into the outside world with the contents of the stomach or intestines. They change into the chrysalis stage, either in the ground or in horse manure. The flies creep out after thirty or forty days and after copulation the females deposit their ova on horses.



Fig. 51. Ovum with larvæ of *Gastrophilus equi*. Figures to the right double magnification, to the left highly magnified.

The body of the bot-fly larvæ is oblong, pointed in front, rounded off behind; it is composed of eleven rings more strongly curved on the dorsal aspect; the anterior rings are supplied with fine spikes at their dorsal margin. The first segment is supplied with two chitinous mouth-hooks, and above them two buttonlike antennæ or feelers. The full-grown larvæ are up to 20 mm. long and reddish to yellowish-gray in color.

The larvæ of the following species of *Gastrophilus* occur in the stomach of the horse:

1. *Gastrophilus equi* (large stomach bot-fly): A fly twelve to fourteen mm. long which lays its eggs preferably on the anterior portion of the body of the horse. The larvæ adhere to the esophageal portion of the stomach. This fly is very widely disseminated and most larvæ occurring in the stomach of the horse belong to this species.

2. *Gastrophilus haemorrhoidalis* (rectum bot-fly): Somewhat smaller, dark brown, likewise widespread bot-fly; the females deposit their ova preferably on the lips on the tactile hair of the horse, also frequently on other parts of the anterior portion of the body. The larvæ are somewhat smaller, dark-red and they are parasitic on the pyloric portion of the stomach of the horses, also in the duodenum; after they are fully developed and shed, they remain for some time in the rectum, where they assume a green color, after which they are voided.

3. *Gastrophilus pecorum* (cattle bot-fly): Twelve to fifteen mm. long, dark brown. The larvæ are particularly common in Hungarian and Russian horses; they are thirteen to fourteen mm. long, of a dark brown color, are preferably found in the stomach and duodenum and, after full development, remain in the rectum for some time.

4. *Gastrophilus nasalis* (nose bot-fly): Light chestnut brown, twelve to thirteen mm. long, more rare than the preceding species. The yellowish-white larvæ

lying tissue from the thickened epithelial covering. There is a proliferation of the gastric glands and microscopically small adenomalike formations subsequently develop in the tissues below the base of the ulcer. After the detachment of the larvæ cicatricial nodules remain in the mucosa.

**Symptoms.** Aside from those very rare cases which go on to perforation, the invasion of a larger number of *Gastrophilus* larvæ leads, particularly in foals, to symptoms similar to those of strongylosis in lambs (stomach-worm disease of lambs, Kröning). According to the intensity and the time of the infection and the nutritive condition of the affected animals, the symptoms come on either towards the end of the period of pasturing or shortly after the stabling of the animals, or occasionally only during winter. In the foreground of the clinical picture are variable appetite, poor appearance of the animal, pale discoloration of the mucosæ, marked emaciation and occasionally symptoms of colic. The heartbeat becomes thumping, the pulse weak, the general condition poorer and poorer, and the animals, which finally suffer from a complete lack of appetite, have dwindled down to mere skeletons, so that they finally cannot get up from the floor any longer. If proper treatment is not instituted the animals may die from six to eight week after an intense invasion (Kröning) or perhaps only after two to four months; in milder cases spontaneous recovery occurs. Kater observed in a horse the picture of stenosis of the duodenum; Tegg that of pylorus stenosis; Rexilius saw increasing cachexia, lack of appetite and increased thirst.

The presence of larvæ of *Gastrophilus hæmorrhoidalis* and *G. nasalis* causes an irritable condition of the rectum, which leads to symptoms of gastric catarrh (frequent defecation, straining, itching, eventually excitement and restlessness, similar to those in colic); straining may exceptionally lead to prolapse of the rectum.

**Diagnosis.** The greatest significance must be attached to the history of the case (onset of the disease shortly after a rather long period of pasturing) with a negative result of the examination of the various organs. Additional evidence is forthcoming if *Gastrophilus* larvæ are expelled from time to time per rectum and if such larvæ are found attached to the rectum. Cachexia of foals (see page 342), can be excluded by the fact that it disappears entirely during pasturing or at least improves considerably. The presence of ascaris in the intestines can be ascertained by finding the worms or their ova in the feces. The exclusion of sclerostomiasis is always very difficult and often can be made only on postmortem examination; both diseases may occur simultaneously. Infectious anemia attacks horses without difference of age and, in chronic cases which alone have to be considered with reference to differential diagnosis from *gastrophilus* infection, causes repeated attacks of fever with little disturbance of appetite.

**Treatment.** The ordinary anthelmintic drugs have no effect upon the very resistant gastrophilus larvæ. The larvæ, however, are killed or narcotized by carbon disulphide (carbonyl sulphide) which has been recommended by Peroncito and Bosso and they are then voided with the feces. The drug is administered in gelatin capsules of 12-15 gm. (foals only 6-8 gm.) three or four times at intervals of two hours, after the animals have been starved for twelve to twenty-four hours. The medicine is administered with the aid of a balling gun or the capsules may have been lubricated with castor oil or vaselin and they are placed on the tongue by the hand, provided the animals are not too young; they are then swallowed by deglutition due to reflex irritation. One may improvise a simple balling gun by splitting crosswise one end of a reed 12-15 cm. long; the four sections made by the cross incisions must be rounded off. One end of the capsule, made slippery as indicated above, is grasped between the four sections of the reed and is pushed along the hard palate, back to the velum, then the capsule is swallowed (Tarr). After twelve to twenty-four hours, one administers 250-500 gm. of castor oil, or tartar emetic (6-10 gm.) with sweet milk or mucilage or an aloe pill (15-30 gm.). The efficiency of carbon-disulphide has frequently been confirmed (Bugarli, Cognesi, Hanke, Wessel, Kröning and others); the discharge of the larvæ begins as early as the next day; they are found in the feces in various stages of development. Animals which are greatly emaciated are much affected by energetic treatment and some may even succumb to it (authors' observation). Santy found oil of turpentine effective (50-80 gm.); it is given in milk.

Larvæ in the rectum may be removed manually or by injections of soap-, vinegar-, or creolin-water; also with carbon-disulphide (5 to 10 gm. to one quart of mucilage). Labat recommends the inunction of the rectal mucosa with boro-vaselin, whereupon the larvæ become detached, without dying, and are voided. The discharged larvæ should be destroyed.

**Literature.** Kröning, Z. f. Vk., 1906, 202 (Lit.).—Petit & Germain, Bull., 1907, 405.

**Nematodes in the Stomach of the Horse.** The fine filiform Spiroptera megastoma forms firm tumors up to the size of a hen's egg in the neighborhood of the left margin of the glandular mucosa; the worms can be expressed from the opening of the tumor. This worm as well as Spiroptera microstoma, infect, according to Railliet, the gastric mucosa of horses and produce in donkeys even ulceration of the stomach; they as well as Strongylus tenuissimus, found once by Mazzanti in the stomach of a horse, are of no significance from a clinical standpoint. Weston saw, however, several cases of ulceration in the pyloric portion of the stomach or in the duodenum due to Sp. megastoma with subsequent peritonitis and death. Petit & Germain saw adenomatouslike formations in the stomach of a horse due to the presence of Strongylus Axei.



(b) **Stomach-worm Disease of Sheep and Goats. Strongylosis ventriculi ovum et caprarum.**

**Occurrence.** The disease occurs in marshy territories, exposed to frequent rains and inundations, and it sometimes causes great losses. Animals of all ages are affected, but preferably only lambs, kids and yearlings. The affection is frequently seen simultaneously with the lung-worm-, or the liver-fluke disease.

**Etiology.** The disease is caused by **strongylidæ**, most commonly by *Strongylus contortus*, more rarely by *Strongylus filicollis*, *Strongylus vicarius*, *Strongylus circumcinctus*, *Strongylus retortæformis*, *Strongylus Ostertagi* or by other species of *strongylus*.

*Strongylus contortus* is a filiform worm one to two cm. long (females two to three cm.); its red color is believed by the majority of authors to be due to blood coloring matter obtained from the stomach; others, however, including Lignières, think that the coloring matter is not hemoglobin.

The embryology of the stomach strongylidæ has not been definitely settled; the investigations of Ransom, Piana and Stödter have shown, however, that the ova of *Strongylus contortus*, *Strongylus Ostertagi* and *Strongylus retortæformis*, voided with the feces of infected animals, under favorable conditions of temperature, discharge embryos which obtain their nutrition from the excreta in which they were set free and grow up to the size of about one mm. If the larvæ are then taken up by sheep they attain their full length after a sojourn of two to three weeks in the abomasum of these animals.

The resistance of the larvæ is very great. According to Ransom they can stand desiccation for thirty-five days, and according to Piana up to ten months. They become immobile in much water; can, however, revive later on. They can stand the cold of winter without any harm and remain alive on pastures for seven or eight months.

**Natural infection** occurs during pasturing by the ingestion of plants contaminated with *Strongylus* larvæ, possibly also with the drinking water. Lambs are said sometimes to become infected during barn feeding (Michalik). Eggs as well as embryos up to their fourth to fourteenth day of embryonic life are not infective (Ransom).

**Pathogenesis.** The strongylidæ bore into the gastric mucosa and suck blood from it; in this manner they disturb the nutrition of the hosts in a degree proportionate to their number. More detrimental than the loss of blood is, however, probably the absorption of toxic metabolic products (according to Grosso hemolysins) of the parasites.

The injuries produced by the parasites afford an opportunity for bacterial invasion. Lignières showed that a disease prevalent in Argentine and known under the

**Prophylaxis.** This consists in avoiding wet, marshy and suspicious pastures and the separation of the sick and of all adult animals from the lambs and kids. The animals should have access to good and, if possible, to running water; eventually there should be elevated stone steps leading to the source of water to prevent the animals from tramping into it or contaminating it with their feces (Stödter). If a continuous avoidance of infected pastures is not possible, they ought to be abandoned as such for one or two years and ought to be utilized for other purposes. Ransom recommends a procedure in this respect which promises permanent results.

From October to March the animals may be kept in a common barn without consideration as to age, and they may be pastured together in April in a non-infected pasture. In May the pasture is changed every two weeks, and in June every tenth day, and between July and August every week in such a manner that the preceding pastures are no more visited during the same year; in September the change of pastures should again occur in longer intervals. Next year the same pastures may again be visited in the same order of rotation, because the disseminated ova or embryos have perished in the mean time. In the fall the adult sheep should receive an anthelmintic course of treatment.

Another method consists in dividing the pasture by a small neutral strip into two portions, one for the lambs and one for the adult animals. The lambs should be allowed to be with their mothers only during sucking. If covering of the females is so arranged that ewing occurs during the winter months, the lambs may be separated from the ewes at the beginning of pasturing and they can then be pastured in localities which have not been visited by sheep for one year.

**Literature.** Michalk, B. t. W., 1891, 573.—Piana, Clin. vet., 1906, 15.—Ransom, Vet. Journ., 1907, 340.—Stödter, Die Strongyliden im Labmagen der gazähmten Wiederkäuer und die Magenwurmsuche, Dis. Bern., 1901 (Lit.).

### (c) Stomach-worm Disease of Cattle. *Strongylosis ventriculi bovis*.

**Occurrence.** Strongylidæ are frequently parasitic in the abomasum of cattle, but they produce disease only if present in large numbers. The disease is then usually seen in young cattle after they have been pastured. Sometimes the disease occurs in an enzootic form (Harker, Penberthy, MacFadyean, Liéniaux, Klein).

In the Berlin abattoir, Ostertag found *Strongylus convolutus* (Str. Ostertagi, Stiles) in 90% of the cattle killed. According to Stödter the stomach strongylosis of cattle is found everywhere in the world.

Schnyder, who has studied stomach strongylosis, claims that the disease known in the country around the Zürich Lake as "*Kaltbrändigkeit*" is a strongylosis found during all seasons and in cattle of all ages, independently of dry or green feeding. He could demonstrate the presence of the disease in 0.2% of all cattle. Bang, however, calls attention to the fact that these cases, and others frequently observed in cattle, may be a combination of gastric strongylosis with the much more dangerous enteritis paratuberculosis Bang (see Vol. I).

**Etiology.** According to Schnyder's investigations there occur in the abomasum of cattle *Strongylus Ostertagi* Stiles (Str. convolutus), Str. retortæformis of Zeder, Str. Curticei Giles (Str. ventricosus), Str. oncophorus Railliet, Str. filicollis Rudolphi and the Str. contortus Rudolphi. One usually finds several species simultaneously in the stomach and intestines of cattle.

ferential diagnosis has to consider particularly enteritis paratuberculosa (see Vol. I).

**Treatment and Prophylaxis.** In cases not very much advanced Schnyder had good results from the administration of flores cinæ (150-200 gm. prepared as a decoction). One may also use the drugs mentioned under worm disease of sheep; the doses have to be increased proportionately (see page 458). Klein obtained favorable results with creosote.

The prophylaxis calls for measures like those recommended against strongylosis of sheep (see page 458).

**Literature.** Blunschy, Unters. über d. Veränd. d. Schleimh. b. d. Magen-Darm-Strongylose, Inaug. Diss., 1906 (Lit.).—Klein, Vet. Jahrb., 1906, 131.—Liénaux Ann., 1900, 438.—Ostertag, Z. f. Flhyg., 1890, I, 1.—Schnyder, Beitr. z. Kenntn d. Magen-Darm-Strongylose, Inaug. Diss., 1906 (Lit). \*

**Stomach-worm Disease in Roes.** Roes are sometimes infected and many may die in consequence of invasion by *Strongylus contortus*, *Strongylus Ostertagi* and *Strongylus filicollis*. In more intense invasions the affection leads to hydremic cachexia (Feser, W. f. Tk., 1903.—Stroh, Z. f. Flhyg., 1905, XV, 163).

#### (d) Parasites in the Stomach of Swine.

The following nematodes occur in the stomach of hogs:

1. **Spiroptera strongylina**; a slender whitish worm, males 10 to thirteen mm., females 12 to 20 mm. long; it forms small tumors in the submucosa; it bores into the mucosa and may in this manner cause serious gastritis. This parasite has repeatedly caused enzootics, terminating fatally within three to four days (V. Ratz).

2. **Gnathostoma hispidum** (*Cheiracanthus hispidus*); cylindrical worm, 2 to 3 cm. long, thickened at the anterior end, adhering to the gastric mucosa or boring into it, the posterior end projecting free into the lumen. The worm produces intense inflammation of the mucosa, thickening of the gastric wall, dilatation of the stomach, disturbances of digestion, cachexia (Csokor); the parasite was found in Hungarian hogs by V. Ratz and Stroese.

3. **Simondsia paradoxa**; found in England by Simonds, in Hungary by V. Ratz in the stomach of swine; the females, 45 mm. long, were seen inside of cysts of the gastric wall, projecting with their head into the cavity; the males are found free in the gastric contents.

4. **Strongylus rubidus**; This worm was found by Hassall and Stiles in 25 to 75 per cent of all hogs examined in the stockyards of Washington, D. C.; Oppermann saw an enzootic outbreak of the disease due to this worm among breeding sows of a farm in Westphalia, Germany. The infection occurred while the animals were in an unpaved yard continually contaminated with manure. The worms produce diphtheroid chronic inflammatory changes in the gastric mucosa, which lead



gering; they have dull eyes at the end of the first day; open their bill wide and succumb after being sick one day; occasionally almost immediately after the onset of the first symptoms.

For prophylactic purposes the sick animals should be killed and ducks and geese ought to be kept away from the suspicious water. During the months of June and September the latter is usually yellowish or brownish due to the presence of numerous small crabs.

Rust saw an enzootic among young ducks, infected in their glandular stomach with *Tropisurus fissispinus* (*Tropidocerca fissispina*); the parasites had produced ulcerative changes of the gastric mucosa. The infection had been produced likewise by *Daphnia pulex*.

Freese saw disease in young geese due to the presence of *Strongylus nodularis* under the mucosa, less frequently in the dark-brownish epithelial covering of the muscular stomach. The disease manifested itself in progressive emaciation and feebleness in spite of good appetite, and ended fatally within three to eight days. Some few species of this parasite are frequently found in otherwise healthy geese.

**Literature.** Freese, D. t. W., 1908, 713.—Hamann, Cbl. f. Bact., 1893, XIV, 555.—Nicolas, J. vét., 1904, 136.—Rust, Pr. VI., 1905, II, 30.—Sturhan, Z. f. Vk., 1903, 131.—Wolffhügel, Z. f. Flhyg., 1903, XIV, 13.

## 29. Worms in the Intestinal Tract. Helminthiasis.

Worms parasitic in the intestinal tract produce disease in a variety of ways.

Contact with the mucosa, adhesion to it, boring into it, produce an irritation, which, according to the number of worms, may cause a circumscribed or a diffuse catarrh or even an intense inflammation. Worms armed with hooks may penetrate more deeply into the mucosa; they may even perforate the intestinal wall and cause fatal peritonitis.

Great numbers of larger worms may unite in lumps and may narrow or obstruct the intestinal lumen. On the other hand individual worms may penetrate into a duct opening into the intestines, especially into the bile duct; they may occlude it and prevent the discharge of a secretion or excretion; they may exceptionally get from the intestine into the stomach, esophagus, pharynx, buccal cavity or larynx.

By withdrawal of nutritive material intestinal worms, when present in larger numbers, cause emaciation, eventually also anemia of the host; in the production of the latter the toxins (leucomaines or ptomaines [Linstow]) of the worms play an important rôle.

Poisonous substances have been demonstrated in many helminthes. They produce destruction of the red blood corpuscles, reduction of hemoglobin, anemia with poikilocytosis, the appearance of nucleated red blood corpuscles, including megalo-blasts and an eosinophilia of the blood. They also produce an increased decomposition of the proteids of other organs.

Many intestinal parasites may bring about secondary infections in consequence of injury to the mucosa. Such secondary

of the segments. The canaliculi terminate in an opening on the posterior margin of the last segment.

The **development of tapeworms** occurs as follows: After the ova, which are voided with the segment, have gotten into the stomach of a proper host, their shell is dissolved and the free embryo (oncosphere) perforates the wall of the stomach or intestine, and gets actively or passively into various organs; in case it comes from a tænia it changes into a bladder-worm or cysticercus; while the embryo of *Bothriocephalidæ* forms a band-like pleurocercoid. If the cysticercus or pleurocercoid subsequently gets into another proper host, the scolex becomes extruded, the bladder or the body of a pleurocercoid is digested and the free scolex or pleurocercoid wanders into the intestine, attaches itself to the mucosa and the development of segments begins. Embryos are formed in the ova after fertilization. From time to time, shorter or longer portions of the worm become detached and are voided with the feces (*T. echinococcus* consisting of three or four segments is voided completely), the segments remain outside on the ground, on straw, or grass. They remain alive for a few days, show even some motion, then they die, the ova, however, are protected by a thick shell, and remain alive much longer, provided that there is some moisture. *Bothriocephalidæ* deposit their ova in the intestinal tract of the host.

The morbid effect of tænia is due to a withdrawal of nutritive material, also to their poisons (Linstow); serious disturbances of health are observed only in the presence of numerous tapeworms.

**Literature.** Leuckart, *Die Parasiten d. Menschen*, 1881, I, 340.

### (a) Tapeworms in Horses.

**Etiology.** Three hookless tapeworms occur in the horse:

1. *Anoplocephala (Taenia) plicata*: 10-25 cm. long, flat tapeworm with comparatively large (4 mm. wide) square head, supplied with four strong suckers. The ova are round or cubical.

2. *Anoplocephala (T.) perfoliata*: 2.5-8 cm. long and 3-15 mm. wide; found in the small intestine, exceptionally also in the stomach. Head blunt, 2 mm. thick with well developed suckers, behind the head on the upper and lower side two rounded flaps. The ova are polygonal.

3. *Anoplocephala (T.) mamillana*: Only 1-5 cm. long and 4-6 mm. wide. The oval suckers found on a spherical head are provided with a central furrow-like depression. The ova are elongated. Is found in the duodenum and jejunum.

Tapeworms are comparatively rare in horses; *A. perfoliata* is the most common among them. The cysticercus of these tapeworms and the manner in which horses are infected are unknown.

**Anatomical Changes.** If the intestine contains many tapeworms, its wall bulges out here and there, and the worms are

**Treatment.** The same drugs as those recommended above for the horse may be utilized.

**Literature.** Eggmann, Schw. A., 1894, XXXVI, 10.—Fumagalli, Clin. vet., 1902, 529.—Haubold, S. B., 1888, 72.

### (c) Tapeworms in Sheep.

**Etiology.** In the intestinal tract of sheep are found somewhat frequently only the *Moniezia* (*Taenia*) *expansa* (see above; also in goats not uncommon), and the *Moniezia* (*T.*) *alba*. More rare are *Taenia ovilla* (according to Perroncito, more frequent in Italy; according to Lungwitz, less so in Germany); *Moniezia* *Benedeni*, *M. trigonophora*, *Stilesia centripunctata*, *St. globipunctata* and others.

From a practical standpoint the most important is *Moniezia expansa*, because in summer it may occasionally cause numerous infections in lambs and yearlings, exceptionally also in older sheep, and may even cause numerous deaths (so-called worm epizootics). Lambs born during the winter, most frequently fall victim to this disease if they are driven to the pasture in spring or at the beginning of summer, during moist weather. Although more rarely, the disease may come on during stall feeding, and lambs sometimes sicken when they are still very young. Spinola found tapeworms 10 meters long in a lamb of four weeks; this points to a very rapid development of the *tænia* since infection can have occurred only after the birth of the lamb.

**Symptoms.** The symptoms of infection are primarily only disturbances of digestion that are not very marked in character; later on some animals of a herd are less lively, they separate from the herd, become emaciated and remain stunted in development. The mucosæ of such animals are found pale, the wool is dry and can be pulled out easily. The animals occasionally exhibit symptoms of colic, strain without voiding any feces and then run away with their tail elevated. Later on the feces become mushy or even thin fluid, and in them may be recognized the yellowish white, motile proglottides. Occasionally obstruction of the small intestines by masses of worms occurs, death then usually ensues rapidly, even before the emaciation has been fully developed. (Blumenberg saw numerous fatal cases, due to intestinal obstruction of this kind.)

**Treatment and Prophylaxis.** It is advisable to give green feed to the animals for several days before the treatment proper is instituted, also pine needles or juniper berries. The following anthelmintics are to be recommended: Oil of turpentine, alone or mixed with hartshorn oil (āā teaspoonful), extractum filicis maris aethereum (5-10 gm. with oil), kamala (4-6.0 gm., recommended strongly by Hartmann upon the results of com-



parative experiments), picric acid (0.1-0.2 gm.) and picrate of potash (0.5-1.5 gm. in pills); except kamala, these drugs should be combined with, or followed in 2 to 3 hours by laxatives (0.2-0.5 tartar emetic, castor oil in tablespoonful doses). This course is to be repeated after 1 to 2 weeks.

Prophylaxis consists in keeping the sheep, especially the lambs, away from infected pastures.

**Literature.** Blumenfeld, Vet. Jhh., 1905, 154.—Hartmann, Mag., 1862, 123.—Lungwitz, A. f. Tk., 1895, XXI, 105.—Marotel & Moussu, J. vét., 1906, 223.—Neumann, Rev. vét., 1891, 251.

#### (d) Tapeworms in Dogs.

**Etiology.** Five *tænia* are more commonly found in dogs:

1. *Taenia serrata*: 0.5-2.0 meters long. Head small, supplied with a wreath of hooks 34 to 38 in number. Segments about 400, in the middle of the chain square, more distally they are elongated, rectangular (8-10 mm. by 4-6 mm. wide); the posterior margin projects laterally, so that the lateral margin of the chain is serrated. The uterus has 8-10 branches on each side. Ova oval 36-40  $\mu$  long; 31-36  $\mu$  wide.

The larval stage, *Cysticereus pisiformis* develops in the omentum and mesentery of rabbits and hares and with these organs usually gets into the stomach of hunting dogs. After two months mature proglottides are then voided. Rabbits and hares infect themselves from plants contaminated with the feces of dogs.

Lesbre found *Cysticereus pisiformis* in the brain of a dog infected with *T. serrata*, where it must have gotten by auto-infection (Friedberger & Fröhner).

2. *Taenia marginata*: 1.5-2.0 meters long. The small head is provided with a double wreath of hooks (30-44) and has four round suckers. The posterior wavy margin of the segments overlaps partly the following segment, the mature proglottides elongated, 14-16 mm. long, 5-7 mm. wide; uterus on both sides provided with 5-8 times dividing branches. Ova somewhat oval, diameter 31-36  $\mu$ .



Fig. 53. Cocon of *Dipylidium caninum*.

This *Tænia* develops from *Cysticereus tenuicollis*, which is found on the peritoneum, the capsule of the liver, exceptionally also on the pleura and the pericardium of herbivora, particularly of sheep and hogs. Butchers' dogs are particularly exposed to the infection, these animals void mature segments 4-5 months after infection.

3. *Taenia coenurus*: It is rarely longer than 40-60 cm., head small, with four suckers and a wreath of 22-23 hooklets. The segments of the median portion are square, the caudal segments elongated; 8-16 mm. long, 3-4 mm. wide. The mature segments are like cucumber seeds. The uterus has 13-25 branches on both sides, the ova are somewhat elongated with a diameter of 31-36  $\mu$ .

The larvae of *Cœnurus cerebralis* develop in the brain, occasionally also in the spinal canal of the sheep, rarely in other herbivora. These infected parts are frequently eaten by butchers' or shepherds' dogs, and these then develop several *Tænia* in their intestinal tract, because each bladder contains numerous proscollices. Mature segments are voided after 2 to 2½ months.

4. *Dipylidium caninum* (*Taenia cucumerina*) is 10 to 40 cm. long, 3 mm. wide at the utmost. The small head terminates in a rostellum which can be projected and withdrawn between the poorly developed suckers. The rostellum is provided with about 60 hooks, arranged in four rows. The mature segments are pale red, shaped like a pumpkin seed, and show a sexual pore on each side. Several ova are united to a sticky mass, a so-called cocon (fig. 53).

According to Melnikow the cysticercus of this *Tænia* (*Cryptocystis trichodectis*) is found in the hair parasite of the dog, *Trichodectes latus canis*, and according to the more recent investigations of Grassi in the dog flea *Pulex serraticeps*, also in the flea of man, *Pulex irritans*. Dogs infect themselves by ingesting these parasites; even sucking puppies may do so (parasites 2.5 cm. long were found in a puppy 10 days old, and mature *Tænia* in puppies 1 to 2 months old).

5. *Tænia echinococcus*. Three or four segments, not over 5 mm. long. Scolex provided with four suckers and a rostellum of 28 to 50 small hooks, arranged in two rows. The second and third segments are incompletely developed and the fourth only is sexually mature. The embryo remains alive in the ovum when desiccated for 10 or 11 days, in water 10 to 16 days.

This *Tænia* develops from *Echinococcus polymorphus* found in the internal organs, preferably the liver and the lungs of ruminants, hogs, also of man. After the ingestion of such infected organs by dogs there develop usually numerous *Tænia* in the intestines of these animals, since every bladder ordinarily contains several hundred protoscolices. *Tænia* develop from these in 6 or 7 weeks. Different species of *Tænia echinococcus* are developed from *Echinococcus cysticus* and *E. alveolaris*, according to claims of Ostertag, Müller, Mangold, and Possell.

Much rarer than those enumerated are *Tænia lineata*, *T. serialis*, *T. Krabbei*.

In regions where dogs have occasion to eat fish they also harbor *Bothriocephalus* (seen in Hungary by v. Ratz). The ova of this intestinal parasite set embryos free in water, which are supplied with cilia that wander into fishes (perch, pike, lake trout, brook trout) to infect their intestinal muscularis or other organs as band-shaped pleurocercoids. If taken up with the infected fish they soon develop in the intestinal tract of dogs and form mature segments after four weeks. *Bothriocephalidæ* are flat, the head is elongated with two longitudinal furrows, the surface of the mature segments shows the rosette-shaped uterus which open up on the surface.

The most common representative is *Bothriocephalus latus* which is found particularly on the shores of large bodies of water, also in man. This worm is 2 to 7 meters long in dogs; the head is lancet shaped, the mature segments are 4 to 6 mm. long and up to 2 cm. wide. After the ova have been deposited the segments shrink (mature segments are not detached like those of *Tænia*). Ova oval 68 to 71  $\mu$  long, 44 to 45  $\mu$  wide; they have a lid at one pole which can be well seen after treatment with sulphuric acid (fig. 54).

Other *Bothriocephalidæ* are found in Iceland and Greenland (*b. curdatus*, *b. fuscus*).

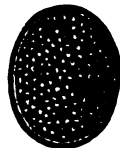


Fig. 54. Ovum of *bothriocephalus latus*.

Concerning the occurrence of various *Tænia* in dogs, the following may be said:

*Dipylidium caninum* is found most frequently, because dogs infect themselves easily by the fleas and lice found in their coat. Butchers' dogs infect themselves easily with *Tænia echinococcus*, *T. marginata* and *T. cœnurus*; shepherds' dogs particularly with *T. cœnurus*; hunting dogs with *T. serrata*. The frequency of the occurrence of these *tænia*s also depends, a good deal, upon external circumstances. Tapeworms in dogs are rarer in large cities with well regulated slaughter houses, with strict meat inspection, and more common in the country where the supervision is less rigid. Young dogs are more susceptible to infection than older animals.

There may be several species of *Tænia* in one host. The

sibly even decreased. Sometimes attention is attracted to infected dogs, when they exhibit signs of itching in the anal region, and either lick it or slide on their haunches.

One may find proglottides in the feces, in vomited matter or in the region of the anus; ova of *Bothriocephalus* are found in the feces, occasionally also ova of *tænia*. Laxatives ought to be given in doubtful cases, because these generally expel segments of *tænia* if present; however, *T. echinococcus* is frequently overlooked on account of its small size.

**Treatment and Prophylaxis.** After starving the animal for one day and cleaning out the intestine by mild laxatives or infusion of water, the vermifuges are administered. In order to prevent vomiting the animals should first receive some mucilaginous soup. It is sometimes necessary to introduce the vermifuge with the stomach tube, after the stomach has previously been anesthetized; for this purpose one may use a mixture of 0.1 gm. of cocaine, 1.0 of a 0.1% sol. of suprarenin with 10.0 aqua amygdal. amar. (10-30 drops), or anesthesin (0.5 gm.) (Uebele). The vermifuges to be used are: Kamala (2-8 gm. in milk, honey or syrup), the last, besides being a reliable anthelminthic, acts as a laxative. Extractum filicis maris (0.5-5.0 gm.) in pills, or better in gelatine capsules; 1 to 2 hours later, 30-50 gm. castor oil must be administered, or the drug may at once be applied in castor oil. Gmeiner recommends as very effective filmaron (0.2-0.4-0.7-1.0 gm. in gelatine capsules or as oil of filmaron); when this drug is used the animals should not be starved; this precaution is necessary to prevent poisonous effect of the drug; one hour after its administration castor oil should be given. Flores Kusso (3-5.0 gm. in milk or water are given every hour, repeated 2-6 times); arecanut (5-10 gm.) with butter in keratinized pills; if there is no spontaneous defecation within two hours, castor oil is to be administered; according to Schiel the dosage is as follows: 10 gm. kamala and 20 gm. arecanut are rubbed up with cocoa butter into 25 pills and these are coated with keratin and given in doses of 6 to 15 pills at a time. Regenbogen recommends Bengen & Co.'s worm capsules; these contain 1 gm. arecanut and 1 gm. of kamala and castor oil (5 pills for large, 1 pill for small dogs). Cortex punicæ granati (5-50.0 gr.) is macerated for 24 hours in a quart of water; this is then boiled down to one pint and given in three fractional doses at intervals of one hour; three hours after the last dose castor oil is administered. As home medicines are recommended, pumpkin seeds (25-50) or turpentine (2-4 gm. with the yellow of an egg, repeated on several consecutive days).

Prophylactic measures are the prevention of any chance for the dogs to feed on infected organs, and proper cleanliness of the coat to destroy fleas and lice.

**Literature.** Dobbertin, Über d. Verhalten d. weissen Blutkörperchen usw. Inaug. Diss., 1907.—Gmeiner, D. t. W., 1907, 514.—Mangold, Über den multikolären



*Echinokokkus* und seine Tænie, Inaug. Diss., 1892.—Müller, Münch. m. W., 1893, 241. —Neumann, Rev. vét., 1891, 417.—Fosselt, Münch. m. W., 1906, 537, 605 (Lit. on *T. echinocoecus*).—Railliet, Bull., 1895, 197.—v. Rätz, A. L., 1904, 41.—Regenbogen, B. t. W., 1908, 425.—Schiel, B. t. W., 1901, 648.

### (e) Tapeworms in Cats.

**Etiology.** Among the tapeworms parasitic in the intestinal canal of cats, the most important is:

*Taenia crassicolis*, a tapeworm 15 to 60 cm. long, with a globular head with four large, prominent suckers and a strong rostellum, which carries a wreath composed of 29 to 52 hooklets. Immediately after the head follow segments as wide or wider than the former; these segments increase in size from 8 to 10 mm. long and 5 or 6 mm. wide. The ova have a diameter of 21 to 27  $\mu$ .

The tapeworms develop from *Cysticereus fasciolaris*, which is contained in the liver of mice or rats.

Cats are also infected by the following other tapeworms: *Taenia elliptica* (probably identical with *Dipylidium caninum*) which may be present in several hundred individuals in the same host; *Dipylidium Chyzeri*, the rostellum of which is provided with rose thorn-like hooklets arranged in 12 to 13 rows; further, *Taenia lineata* and *Bothriocephalus felis*.

**Symptoms.** *T. crassicolis* bores deep into the wall of the intestine and may exceptionally perforate it. If these tapeworms are present in larger numbers they may cause chronic intestinal catarrh, with poor appetite, diarrhea, then constipation, salivation, diminution of the visual and auditory power, emaciation, finally convulsions. The disease sometimes appears in an enzootic form, particularly in years when mice are very abundant.

**Treatment** is the same as in dogs, but the vermifuges, etc., have to be given in one-half the doses indicated for dogs.

**Literature.** Deich, S. B., 1901, 46.—V. Ratz, Közl., 1897, II, 28, 38.

### (f) Tapeworms in Rabbits.

Hookless tænia are rarely found in rabbits, frequently in wild hares, they are distinguished from the *Anoplocephalidæ* of equidæ by an alternate arrangement of the genital pores on the margin of the proglottides. These tapeworms are comprised under the name of *Taenia pectinata*; according to Railliet and Neumann, they represent three distinct species.

Tapeworm infection frequently appears enzootically among wild hares, rarely in such wide distribution among rabbits; its clinical symptoms are a progressive anemia with emaciation and increase in the size of the abdomen. Sometimes tapeworms get into the free peritoneal cavity of the infected animals without leaving any distinct traces of perforation of the intestines (Railliet). The diagnosis of this infection can be made from finding proglottides in the feces or from postmortem examination of the animals.

The treatment consists in a preliminary administration of castor oil (tea to tablespoon doses) followed by the following vermifuge which

is to be given in gelatine—or keratin capsules: extractum fil. maris (0.5-1.0 gm.), arecanut (0.5 to 1.0 gm.), kamala (2.0-3.0 gm.), tartar emetic (0.05-0.1 gm.).

**Literature.** Braun, Kaninchenkrkht, 1907, 12.—Lucet, Rev., 1897, 633.—Neumann, Maladies parasitaires, 1892, 461.—Railliet, Zool. Méd., 1895, 283.

### (g) Tapeworms in Fowl.

**Etiology.** In barnyard fowl, in pheasants and in other wild fowls of allied genera, numerous species of tapeworms are comparatively frequent and not uncommonly cause enzootic affections with fatal cases, particularly in young animals. The following and other tapeworms occur in the various species of fowls:

In **chicken**: *Tænia* (*Drepanidiotænia*) *infundibuliformis* (frequent; its cysticeroid lives, according to Grassi, in the house fly); *Davainea* (T.) *cesticillus* (likewise common); *Dicraniotænia* *sphenoides* (according to Grassi and Rovelli its cysticeroid lives in the angleworm); *D.* (T.) *tetragona* (its cysticeroid lives in snails); *Davainea* (T.) *proglottina* (its cysticeroid lives in snails); *Dicraniotænia* (T.) *echinobothrida*; *Bothriocephalus* *longicollis* and others.

In **geese**: *Drepanidiotænia* *lanceolata*; *D.* *setigera*, *D.* *fasciata*.

In **ducks**: *Drepanidiotænia* *anatina*; *D.* *gracilis*; *D.* *sinuosa*; *D.* *megalops*; *D.* *coronula*; *D.* *conica*; *Mesocestoides* *inbutiformis*. The larvæ live in water arthropodes.

In **turkeys**: *Drepanidiotænia* *cantaniana*.

In **pigeons**: almost exclusively *Davainea* (T.) *crassula*.

**Symptoms.** As a rule the presence only of numerous tapeworms brings about morbid symptoms in fowl. The animals are then less lively, without appetite, or they eat, on the contrary, quite a lot of feed, but they emaciate in spite of this; later on there is diarrhea, and the animals then become completely exhausted. There are also cases when death comes on quite unexpectedly and where a postmortem examination shows the presence of numerous tapeworms; in other cases there are attacks of dizziness, epileptiform convulsions, caused by marked inflammation, obstruction or perforation of the intestine.

In a tapeworm epizootic among geese imported from Russia, Cämmerer noticed nervous symptoms aside from emaciation and diarrhea. The animals became feeble and their movements awkward; they kept on sitting quietly from the 4th or 5th day of the disease, and permitted themselves to be caught without making any effort to escape. On the 7th day they assumed a position like penguins, with the head immobilized and directed to one point or resting upon the sternum. From time to time the animals made an effort, fell, however, upon their abdomen and then remained in this position or on their side, or they again rose by the aid of their wings into the penguin position.

**Diagnosis.** Tapeworm disease can be reliably diagnosed only by finding the proglottides in the feces, otherwise postmortem examination only can demonstrate the cause of the affection.

**Treatment.** The treatment consists in the administration of vermifuges, which are best given in the form of pills. The most appropriate are: arecanut (pigeons 1 gm., chickens 2 gm., geese 4 gm., young animals 1-3 to 1-2 of those doses); this

drug easily leads to symptoms of poisoning in turkeys; kamala in the same doses as the preceding vermifuge; Ellinger found kamala dangerous for geese: flores kusso (1-3 gm.); sulphate of copper (blue copper vitriol in 2% solution 10-20 drops, or a solution of 1-5 to 1000 given as drinking water); oil of turpentine (0.25-1.0 with mucilage or oil). Seeds of pumpkin may be given to chickens. All these drugs, however, sometimes fail on account of the small size of fowl tapeworms, and on account of their protected situation deep down in the cecal pouches.

As a prophylactic measure the droppings of sick birds should be swept together daily and burned or buried; water fowl should be kept away from infected bodies of water; or one might, perhaps, attempt to kill the intermediary hosts by the addition of disinfecting substances (lime) to the water.

**Literature.** Blanchard, Bull. de la soc. zool., 1891.—Caparini, Clin. vet., 1906, 841.—Fuhrmann, Cbl. f. Bakt., 1909, XLIX, 94.—Klee, Vet. Jhb., 1905, 363.—Poenaru, Arh. vet., 1906, 279.—Stiles & Hassal, Vet. Jhb., 1897, 196.—Wolffhügel, Beitr. Zur Kenntnis d. Vogelhelminthen. Inaug. Diss., 1906 (Lit.).—Zürn, Z. f. Tm., 1898, II, 447 (Ref.).

## B. Trematodes.

(*Saugwürmer* [German].)

Trematodes are hermaphroditic single worms in the shape of a leaf or tongue, with an intestinal tract ending blind, and with organs for attachment. The latter as a rule consist of a mouth sucker and a more caudally situated abdominal sucker.

Few trematodes live in the intestinal tract of domestic animals, and these few species are rarely found; they are of no clinical significance in the temperate zone. The following are the intestinal trematodes so far observed:

1. *Amphistoma collinsi*, redworm, found in large numbers in India in the large intestine of horses and causing serious disease.
2. *Gastrodiscus ægyptiacus*, flatworm of the size of a bean, occurs in some parts of Egypt in the gastro-intestinal tract of horses and cattle.
3. *Amphistoma tuberculatum* has been found in the intestinal tract of Indian cattle.
4. *Hemistoma alatum*, 3 to 6 mm. long, leaf shaped, flatworm with two thorn-like projections at the anterior end. Occurs frequently among wolves and foxes, rarely in dogs.
5. *Echinostomum perfoliatum*, 4 to 15 mm. long, reddish with a lancet-shaped body; its wide kidney-shaped anterior end is armed with spikes. Common in water fowl; Generali and v. Ratz have seen it each in one case in the duodenum of a dog.
6. *Megastomum entericum* (*Dimorphus muris*, *Cercomonas seu lamblia intestinalis*) occurs in rabbits, rarely in cats. Sartirana (J. vét., 1905, 550), in a dog dead from a gastro-intestinal inflammation, found these parasites in large numbers in the stomach and intestines.

There are some more trematodes found in fowl, especially: *Monostoma verucosum*, *Distoma oxycephalum* in ducks, chicken and geese; *Distoma dilatatum*, *armatum*, *lineare*, *ovatum*, *commutatum* in chickens, *D. commutatum* also in pigeons, and *D. ovatum* in geese; *Holostoma erraticum* in ducks; *Monostoma attenuatum* in geese; *M. caryophyllinum* in ducks.

Trematodes as a rule are harmless intestinal parasites; however, they sometimes cause digestive disturbances, emaciation and anemia in fowls, which occasionally terminate fatally. The treatment is the same as in tapeworm infection (see page 413).



### C. Roundworms. *Nemathelminthes*.

The body of roundworms is elongated, cylindrical and not segmented. Thread worms (nematodes) and *Acanthocephala* occur in domestic animals. Both are differentiated into separate sexes. The thread worms are generally provided with a well developed intestinal canal, which opens with two stomata at the surface of the body; *Acanthocephala* have no intestinal canal and the anterior end is formed by a protrusible proboscis, provided with hooks.

#### (a) *Ascaris* in Mammals. *Ascariasis*.

**Occurrence.** *Ascaris* and affections produced by them are frequently seen in young animals; older animals are more rarely affected and then generally only by few worms of this type. Animals which can run around free in the barn or on the pasture are especially exposed to infection, while animals which are tied can more rarely find a chance to take up the ova of *ascaris* (Albrecht). Enzootics have been observed among calves of 3 to 5 months (Leibenger, Gasteiger), among young pigs, occasionally among lambs (Vallisneri), foals, also among dogs.

**Etiology.** Roundworms are long, smooth cylindrical worms, with a short head and with three lips, either smooth or armed with teeth around the mouth.

The following worms occur in domestic mammals:

1. *Ascaris megaloccephala*, white or yellowish white, rigid worm; its three mouth-lips are provided with teeth, male 15 to 28 cm. long, its posterior end conical with two skin-like small wings, directed towards the abdominal surface; the female 18 to 37 cm. long, its hind end blunt, straight; the sexual pore is at the anterior portion of the body. Ova globular or somewhat elongated oval, in the feces of the host usually yellowish-brown, not segmented or only in the first segmentation stage; diameter 90 to 100  $\mu$ , surface smooth (fig. 55). The worm lives in the small intestine of the horse and, according to Schimmelpfennig, the mature individuals also suck blood.



Fig. 55. Ovum of *Ascaris megaloccephala*.

2. *Ascaris vituli*, with reddish-white translucent body. Male 15 to 20 cm., female 22 to 30 cm. long, genital pore in the anterior sixth of the body. The diameter of the ova is 75 to 80  $\mu$ , the membrane appears finely wavy. The worm lives in the small intestine, exceptionally also in the abomasum of calves; it is rarely found in adult cattle (Gasteiger believes that the roundworms which he found in calves were *Ascaris lumbricoides*).

3. *Ascaris ovis*, yellowish-white worm, mouth-lips provided with teeth; male 7 to 10, female 7 to 12 cm. long. Very rarely found in the intestine of the sheep.

4. *Ascaris lumbricoides* (*ascaris suilla*), body white or pale reddish, mouth-lips provided with teeth. Male 15 to 17 cm., female 20 to 25 cm. long; the anterior end of the male is bent like a hook, that of the female straight; the female genital

pore is at the boundary between the first and second third, situated in a ring-shaped depression. Membrane of ovum wavy, ova 66  $\mu$  long. Lives in the intestinal tract of swine and man.

5. *Ascaris marginata*, the head of the white or pale reddish body is provided on each side with one oval wing; this makes the worm look somewhat like a lancet; male 5 to 10 cm. long; on the bent posterior portion two narrow wings; female 9 to 12 cm. long, posterior end conical; the genital pore in the neighborhood of the first quarter. Ova globular, diameter 75 to 80  $\mu$ , membrane wavy (fig. 56). This worm occurs in the small intestine of the dog. (Galli-Valerio, in 162 dogs dissected in Mailand, found ascariases in 18.5%.)

6. *Ascaris mystax* is so much like the preceding one that they are now held to be one and the same species; it is, however, considerably smaller than the *Asc. marginata* (4 to 6 or 4 to 10 cm. long; 1 to 1.5 mm. wide). It lives in the small intestine of the cat. (Galli-Valerio found this *Ascaris* in 8 out of 12 cats dissected in Milano.)

The development of *Ascaridae* has been demonstrated by the observations of Grassi, Lutz, Ebstein, Jammes & Martin and A. Albrecht. In order that an embryo in the ova, voided by the female in the intestinal tract of the host, may develop, sufficient access of oxygen is necessary. Hence the development of the embryos can occur only outside of the animal body. When temperature and moisture are sufficient, when free oxygen is accessible and when the reaction of the surrounding medium is acid (Jammes & Martin), then rapid segmentation of the

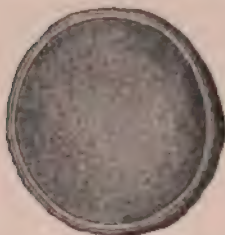


Fig. 56. Ovum of *Ascaris marginata*.

ovum occurs and it terminates in the formation of a lively motile embryo. At a temperature of 37°C., embryos in the ova of *Asc. megaloccephala* (Albrecht) are hatched after 3 to 4 days, at room temperature only after 24 to 30 days; in ova of *Asc. vituli*, embryos are hatched after 18 days if the temperature is near that of the animal body. The development goes on favorably in moderately moist environment, it is prolonged in water, and the latter may bring about partial solution of the yolk. Light has a retarding influence in the beginning stage of development. Ova which already contain embryos appear to be endowed with a particular

power of resistance since they retain their viability for 6 months while kept in desiccated horse manure (Albrecht). Unsegmented ova likewise retain their viability under conditions of cold, however, no segmentation occurs at such low temperatures. The liberation of the embryo never occurs in the external world, only in the alkaline intestinal juice.

The final development of the worms goes on rapidly after the embryos are once in the intestinal tract; 10 to 12 weeks after artificial infection, ova of ascaris may be found in the feces. Some species of ascaris develop even more rapidly, and fully developed worms of this genus have been found in young dogs, two weeks old (Penberthy); Gasteiger has seen clinical signs of roundworm infection in calves 3 weeks old and occasionally in these animals at the age of 10 days.

**Natural infection** occurs through the ingestion of food or water contaminated with ova, with embryos, or by licking objects where ova have been deposited. Ova voided with feces collect on the bedding straw, near watering places, on pastures,

and they sometimes permanently infect the floor of certain barns; from these places they may be taken up by healthy animals or by animals already infected, or they may further spread and contaminate feed, water or other objects in the neighborhood of the animals. Railliet saw an epizootic of ascaris infection in 250 horses, due to the ingestion of infected turf straw. Gasteiger found ova of ascaris in large numbers in the manure and outside of the barn in leaf bedding, where they had been transported by the shoes of attendants and small animals (the infection of the leaves might have occurred in the woods by free living wild animals). Sucking animals usually infect themselves, swallowing ova which have been transported from the bedding straw to the udder or by licking, in a playful manner, straw or manure infected with ova. This may explain the enzootic occurrence of ascaris infection among the calves of Miesbach-Tegernsee, where calves have a strong desire to lick soon after birth (Gasteiger). This mode of infection explains how young animals, which aside from mothers' milk receive no food or water, may become infected with ascaris (Grassi, Fröhner, Penperthy, Albrecht, Leibenger, Gasteiger). If newborn calves suffer from diarrhea (dysentery), the ingested ova are expelled and cannot become parasitic in the intestinal tract (Gasteiger).

Jammes & Martin call attention to the fact that the susceptibility of the individual animals depend upon the chemical composition of the gastric juice, and this again varies according to the condition of the digestive organs, the species, age and general health of the animals. According to the authors named, acid gastric juice, as found in the stomach and possibly in the first portion of the small intestines, favors segmentation, while the alkaline intestinal juice favors the escape of the embryo out of the egg shell.

**Pathogenesis.** Aside from mechanical injury (irritation of the intestinal mucosa, stenosis of the intestinal lumen, possibly rupture) one must, according to Gasteiger, consider the effect of poisonous metabolic products formed by certain enzymes of *Ascaridæ*. These products preferably cause nervous disturbances and inflammatory changes. Mingazzini succeeded in producing convulsions with extracts of *Ascaridæ*, also paralysis, sometimes even fatal affections. Inflammation of the conjunctiva, due to contact of the latter with ascaris, has repeatedly been seen in man (Kitt, Gasteiger). Aside from metabolic products, other matters due to decomposition of the dead worms may, according to Gasteiger, play a certain rôle; this is, perhaps, indicated by the observation that the administration of vermifuges is followed by a short period of increase of the symptoms of ascaris infection. It is also claimed that ascaris excrete fatty acids which impart a peculiar smell to the organs of the host. Nutritive disturbances which occur in the course of ascaris infection are probably mostly due to the abstraction of nutritive material, and to the fact that the mature worms suck blood from the intestinal wall (Schimmelpfennig).



**Anatomical Changes.** At the place of attachment of an ascaris there is formed a small round depression, sometimes covered with blood, in its neighborhood the mucosa shows catarrhal changes. In the presence of numerous worms the small intestine shows inflammatory changes along its whole extent, and deeper ulcerations may be present; perforation of the intestine occurs occasionally, and this is then followed by general peritonitis or the formation of an ichorous-purulent abscess between the folds of the mesentery, or even fatal hemorrhage (Csokor). In the cases of perforation seen in horses, the former always occurred at the place of attachment of the mesentery (Zorn, Kitt, Winz, Budnowski, Franke), because at other points the serosa forms an impediment for the worms (Franke). There is, occasionally, stenosis or obstruction of the intestinal lumen by balls of worms (preferably in dogs and horses), and these may cause rupture of the intestine. Exceptionally roundworms may get into the stomach, the pancreatic or bile duct; they here produce obstruction.

Roundworms produce a peculiarly disagreeable smell in the meat of calves and lambs (Morot, Laubion, Mathis, J. Kunos, Vallisneri).

The number of roundworms is sometimes very high. A report from Brussels gave 1800 as the number of *Ascaris megalocephala* in a horse. Delamothe found 1215 which weighed six and a half pounds. Descamps found a mass of *Ascaris vituli* equal to fifteen liters (quarts). Krabbe 80 individuals of *Ascaris mystax*; Albrecht 250 individuals of *Ascaris marginata* in a dog six weeks old.

**Symptoms.** Animals exhibit morbid manifestations only in the presence of numerous roundworms, the symptoms, however, are not always proportionate to the number of worms. Most marked are the general nutritive disturbances and these are more intense, the younger the animals. The appetite is variable, sometimes entirely suppressed, at other times much increased; in spite of this there is emaciation, the mucous membranes are pale, the coat loses its smoothness and luster. Constipation alternates with diarrhea. Migration of the roundworms into the stomach in cats and dogs causes vomiting, and occasionally one finds the worms in the vomitus, composed of mucus and bile.

Symptoms of intermittent and occasionally violent colic are more frequently seen in horses, more rarely in dogs and cats. Occasionally nervous phenomena occur in horses in the shape of attacks of tetanic stiffness (Dieckerhoff, Hoffmann), also epileptiform convulsions (Trülsen, Dubuisson), maniacal excitement followed by coma (Duncan) and paretic weakness of the hind legs (Danitz). In dogs, worms sometimes cause maniacal excitement. Convulsions without any premonitory symptoms may occur during a meal, in pigs (Bru).

Sometimes the clinical picture of intestinal obturation or of peritonitis set in, or death occurs suddenly without any preceding sickness.

Sick calves and lambs emanate a peculiar, penetrating smell.

Caseiger distinguishes two different clinical pictures in calves. In the mild form one notices a peculiar smell of the exhaled air somewhat like ether, chloroform or alcohol, the urine is light in color, there is cough, but rarely emaciation. In the severe form, aside from these same symptoms, there are disturbances of appetite, constipation or diarrhea, bloating, frequent urination, difficulty in respiration and nervous manifestations (colicky symptoms, listlessness). The duration of the disease is from one to several weeks and in severe cases a stage of convalescence which may last for several weeks.

**Diagnosis.** A reliable diagnosis can be made only after the detection of roundworms or their ova in the feces or occasionally in vomited matter. In suspected cases, vermifuges may be given to secure a diagnosis. However, experienced observers will be able to diagnosticate the disease from the peculiar smell of the exhaled air and of the urine. One may assume the presence of worms in young animals with a good deal of probability if they have previously been healthy, if digestive disturbances of an indefinite nature come on in spite of good attendance, and if in spite of good appetite emaciation develops within a few weeks.

**Treatment.** This varies more or less according to the species of animals. Its success should be controlled by the occasional microscopic search in the feces for ova of ascarides. It is advisable to feed horses previously fairly large amounts of beets or potatoes (8-10 qts. daily with wheat-bran), then numerous worms are, as a rule, expelled (Moebius). The best of the vermifuges is tartar stibiatum; this drug is successful as a rule, though not always (Möller, Cadéac, Albrecht and others). However, this drug is not well adapted for either very young or old worked out animals, because it easily causes collapse in them; sometimes intense colicky symptoms occur after its administration, even in adult animals in good condition (Storch), especially if the drug is given dissolved in water and upon an empty stomach. For this reason Schlampp recommends the administration of tartar emetic (12-15 gm. in 1 qt. of hot water), with 1 to 1½ quart of wheat-bran, the horse having, on the previous day, received half a ration for its noon meal, and nothing at all for the evening meal. Light horses which are much affected by the treatment received, according to Lorenz' method, 30 gm. tartar emetic (dissolved in 1 qt. of water), and this divided into 6 doses, each mixed with bran for 6 successive meals. Schmidt gives 12 gm. of tartar emetic and 50 gm. of sulphate of magnesia, in 2 quarts of hot water; this is given for two consecutive days, in the evening with the drinking water before the evening meal; the animals should not drink any water at all during the day preceding the evening treatment. Instead of arsenic (0.1-0.5 gm.) in



pills or as an electuary, one may give Fowler's Solution for 5 to 8 days, morning and evening, 1 to 2 tablespoonsful for an adult horse or 1 teaspoonful for a foal, in oats, bran, or in the drinking water. *Oleum terebinthinæ* (50-100 gm. with 300 gm. of castor oil) has likewise been found advantageous; also arecanut (for adult horses 100-250 gm., for foals 10-50 gm., or 3 times daily, 1-2 tablespoonsful or 1 teaspoonful with moist short cut feed, bran-mash or as an electuary). Santonin (10-25 gm. in pills or as an electuary), which, however, is quite expensive, may be used for horses, with the exception of very young foals, given in castor oil or followed by the latter. *Carboneum sulfuratum* (carbon bisulphide) is useful against roundworms as it is also against *gastrophilus* larvæ (see page 456). Rehder, who found this drug more effective than anything else, uses it in doses of 50-100 gm. in one dose with 200-300 gm. of castor oil, without any evil consequences.

Gasteiger had good results in calves with the following drugs: Tartar emetic 3-5 gm. dissolved in 125 cc. distilled water; 1 tablespoonful in milk every 3 or 4 hours, until the desired effect is brought about; arecanut with *flores cinæ* (mixed with the feed, calves up to 3 weeks 10 gm.: 5 gm.; up to 5 weeks 15 gm.: 7.5 gm.; over 5 weeks old 20 gm.: 10 gm.). The mixture also makes a very good appetizer. This mode of treatment may also be used in lambs; the doses must, of course, be smaller; or lambs may also be treated with the same drugs recommended against gastric strongylosis (see page 458). For hogs are recommended: Arecanut (adult animals 10-20 gm., young pigs 5-10.0 gm., with honey or flour as an electuary), oil of turpentine (daily 1 teaspoonful in mucilage), picronitrate of potash (0.2-0.5 as an electuary), benzin (10-20 gm., with bran or flour as an electuary). After a few hours laxatives are administered (calomel, aloe, castor oil). Dogs and cats should receive the same treatment recommended against tapeworms (see page 471), also very serviceable are santonin (for dogs 0.05-0.2 gm., for cats 0.02-0.05 gm., in powder, followed by castor oil or still better combined with it), or *pastilli santonini* (each one containing 0.025 gm. santonin), *flores cinæ* (2-10 gm. as an infusion), thymol (0.2-0.5 gm. in pills), and filmaron oil (2-5 capsules at a dose, followed by castor oil).

**Prophylaxis.** The spread of infection may be prevented by separating the sick animals, collecting, burning or burying the feces and drying out the place where the sick animals are kept. Mother animals harboring *Ascaridæ* should be relieved of them before delivery, and kept separated. It is advantageous to examine, microscopically, the feces of larger herds in order to prevent the spread of the disease. Gasteiger recommends, aside from the destruction of the feces, that the floor of the barn be made waterproof, so that it can be swept with iron brooms and can be covered with sand. Before removal, the latter is moistened with



hot lye and then renewed. The udder of the cows must be cleansed carefully and the animals must wear a muzzle.

**Literature.** Albrecht, Z. f. Vk., 1908, 465 (Lit.).—Cadéac, J. vet., 1906, 221.—Damitz, Mag., 1845, 497.—Francke, Fostchr. d. Hyg., 1904, 185 (Lit.).—Gasteiger, Monh., 1905, XVI, 49 (Lit.).—Grassi, Cbl. f. Bakt., 1888, IV, 612.—Hoffmann, Z. f. Vk., 1905, 359.—Jammes & Martin, J. vet., 1906, 604, 607.—Lutz, Cbl. f. Bakt., 1888, III, 425.—Neumann, Mal. parasit., 1892, 387 (Lit.).—Penberthy, J. of comp. Path., 1894, 175.—Schimmelpfennig, A. f. Tk., 1903, XXIX, 332 (Lit.).—Schlampp, Therap. Technik, 1907, II, 40.—Schütt, D. t. W., 1906, 637.—Trülsen, B. t. W., 1893, 14.—Uebele, Therap. Handlexikon, 1910.—Zschokke, Schw. A., 1900, XLII, 254.

### (b) Roundworms in Fowls. *Heterakis*.

**Etiology.** The following roundworms occur in fowl:

1. *Heterakis maculosa* (*Ascaris columbæ*), body white, translucent, head with three large mouth-lips. Male 16 to 25 mm., female 20 to 34 mm. long, both 1 to 2 mm. thick; the former has a sucker on the ventral surface in front of the anus, and two unequally long specula like all *Heterakidæ* in contradistinction to *ascaris*, where the males are provided with two long spicula, but without a sucker. The worm is parasitic in the intestinal canal of pigeons and is sometimes found in several hundred individuals.

2. *Heterakis inflexa*, yellowish worm, always curved. Male 3 to 8 mm., female 7 to 12 mm. long. The male has a roundish sucker and three papillæ on the ventral surface; ova as in *Heterakidæ* always similar to those of *ascaris* (fig. 57). Found in the intestine of chickens and pigeons.



Fig. 57. Ovum of *Heterakis inflexa*.

3. *Heterakis papillosa*, (*H. vesicularis*), slender worm, pointed at both ends, on the anterior portion of the body lateral papillæ. Male 7 mm. long, posterior end pointed, armed with two spicula, unequal in length, female 10 to 12 mm. long. Found in the ceca of chickens, peacocks and turkeys.

The following are found more rarely: *Heterakis dispar* (geese), *Heterakis differens* (in the posterior portion of the intestinal tract of chickens), *Ascaris crassa* (in ducks, according to Diesing, identical with *H. inflexa*); *H. compressa* (in Australian chickens); *H. brasiliensis* (in chickens in South America); *H. perspicillum* (in chickens and pigeons).

*Heterakis maculosa* is the most dangerous among those enumerated above, because it occurs occasionally in large numbers in the intestine of pigeons, and it may cause destructive epizootics. According to Unterberger's investigations the droppings of sick animals contain very numerous ova (in 6 grammes droppings about 1200 ova). Embryos develop in the ova in moist places in the open air within 17 days. If embryos get into the intestinal tract of pigeons, mature worms are found after three weeks. The worms are present in large numbers; they produce enteritis and may cause stenosis or obliteration of the intestinal lumen. Occasionally they develop in the serous cavities without producing disturbance (Sabrazès & Salm).

*Heterakis inflexa* occasionally causes serious disease in chickens, other roundworms are less dangerous and rarer in those animals. v. Rätz saw epizootics and fatal cases in turkeys, and Klee in pheasants, due to *Heterakis vesicularis*, causing inflammation of the ceca. Guittard made similar observations; Railliet & Kasperek saw an epizootic in pigeons and chickens,

2. *Dochmius stenocephalus*, somewhat smaller than the preceding one. The head end pointed, mouth capsule conical, provided with two pairs of small teeth on the ventral side and with a longitudinal furrow on the dorsal side; the bursa of the male similar to that of the preceding species. Male 6 to 8 mm., female 8 to 10 mm. long, ova 63 to 67  $\mu$  by 32 to 38  $\mu$ .

Whether *Dochmius duodenalis*, which causes severe anemias in man, ever occurs in domestic animals is not yet known.

**Development of *Dochmii*** occurs quite rapidly. From ova discharged with feces, rhabditis-like embryos are developed in moist soil within 3 to 6 days; after hatching these develop further in water or moist mud and become encysted. If they then get into the stomach of dogs they mature in the intestinal tract after three moults.



Fig. 58. Ova of *Dochmius trigonocephalus* (according to Railliet).

**Natural infection** is caused by contaminated, marshy water. Numerous ova contained in the feces of infected animals develop when they get into water, and they are quite resistant. One or two sick animals can, therefore, infect a whole pack or a kennel. The disease is usually seen during the warmer season, but it may also make its onset in winter, as a case of the authors' proves. Distemper or any debilitating disease forms, according to Lignières, a predisposing cause and prepares animals for infection. Infection may, however, also take place through the otherwise intact skin, as pointed out with reference to animals by Lafon & Martin, a mode of infection demonstrated experimentally in dogs by Cuillé.

Loos, Lambinet, Calmette & Breton and others have shown that infection with *Dochmius duodenalis* (*Anchylostomum duodenale*) may occur through the intact skin or from the subcutaneous connective tissue.

Cuillé placed encysted larvæ of *Dochmius trigonocephalus* upon the shaved thorax of experimental dogs; after a few minutes the animals manifested excitement by howling and looking around towards the thorax, and rolling. These manifestations were caused by the irritation of the larvæ which had penetrated into the skin. The dogs became quiet after two hours, but the larvæ had in the meantime penetrated into the skin, from where they got into the pulmonary capillaries with the blood current. They then perforated the capillaries and got into the air vesicles, from there into the bronchi, trachea and larynx, and finally into the pharynx, esophagus and stomach. However, it appears more probable that they got into the intestinal wall with arterial blood, and from there by perforation of capillaries into the intestinal lumen.

Edema and erythema, which are first seen at the place of infection, disappear after 4 or 5 days, but on the day following the infestation the animals show an inclination to vomit, they are depressed; after 5 or 6 days they become voracious, suffer from bloody soft stools on the 10th day, and from the 12th to the 18th day ova are found in the feces. Death occurs usually after 20 to 30 days in very young animals, or in 10 days after a very numerous invasion. Larger dogs show the symptoms of progressive anemia but remain alive for a long time.

**Anatomical Changes.** At the point of attachment of the worms one sees smaller or larger hemorrhages, sometimes, also, hemorrhagic intestinal contents. The mucosa of the duodenum and jejunum at first shows the picture of acute catarrh, later on that of chronic inflammation, the intestinal wall becomes less elastic, the lumen may occasionally be narrowed. In animals

just dead, one finds the worms, usually both species, simultaneously, attached to the wall; later on they become mixed with the feces; in severe cases one often sees several hundred parasites. The mesenteric and peribronchial lymph glands are enlarged, according to Lafon and Martin, all of the lymph glands being thus affected.

**Symptoms.** There are at first digestive disturbances of an indefinite nature, then anemia stands in the foreground of the clinical picture. Debility and emaciation become more and more marked, and hunting dogs become tired very easily. The skin becomes dry, the coat bristly, the extremities show edematous swelling. There is at first constipation, later on diarrhea, the feces are occasionally hemorrhagic.

Examination of the blood shows red blood corpuscles, varying in size and containing irregular forms; their number may be decreased so that the proportion of red to white is finally as 3:1. These findings do not, however, justify, in the opinion of the authors, a classification of this disease as pernicious anemia, as has generally been done.

According to the claims of Lafon & Martin, all of the lymph-glands become enlarged early in the course of the disease (before any other symptoms are noticeable) in consequence of the invasion of staphylococci, which are, however, also found in the spleen, the blood and the intestinal contents of animals dead from the disease. It is also claimed that these staphylococci have a hemolytic effect. Another constant symptom is albuminuria (1.5 to 2%, but sometimes as much as 3-8-10% albumen in the urine), together with other symptoms of diffuse nephritis. The albuminuria becomes intensified in consequence of excessive feeding.

French and Italian authors mention epistaxis as an almost constant symptom of the disease, and the disease has received its French name from this symptom (*Saignement de nez des chiens de meute*). Some patients lose 100 gm. of bright red and foaming blood at one time. According to Trasbot the disease characterized by epistaxis is not identical with *dochmiasis*, and Péricard has recently shown *piroplasmata* in the blood of such patients.

While these symptoms occur, the emaciation progresses and death finally follows.

The duration of the disease may extend to one year, and all animals of a pack may become affected successively.

**Diagnosis.** *Dochmiasis* can be diagnosticated with certainty only after finding ova in the feces upon microscopic examination; otherwise, the diagnosis can only be made with more or less probability in hunting dogs suffering from hemorrhagic diarrhea, progressive anemia, general hypertrophy of lymph glands, albuminuria and nephritis. *Pentastomum tænioides* likewise causes epistaxis, but profound nutritive and other disturbances and progressive anemia are absent. The abnormal condition of the feces distinguishes the disease from other forms of anemia.



**Treatment.** The usual vermifuges are employed (see page 471); kamala 3-8.0 gm.; Sequens brought about complete recovery in one case after administering 8 gm.; Mégnin recommends the addition of 0.3-0.5 calomel and 0.005-0.01 gm. arsenic; extr. filicis maris (same doses), and arsenic (0.005-0.01 gm.). The patients must be nourished abundantly with milk, meat and eggs; iron preparations are indicated to counteract the anemia. The treatment is usually not successful, because patients remaining among an infected pack continually reinfect themselves with the *dochmius* larvæ (Lafon & Martin).

**Prophylaxis.** This consists in scrupulous cleanliness. Kennel and food vessels must be cleansed daily, so that the ova cannot get into the food or drinking water. The latter must be fresh and supplied so abundantly that the animals will not be tempted to drink dirty or contaminated water. Sick dogs must be separated from the healthy ones and the feces of the former must be destroyed.

**Literature.** Cuillé, Rev. vét., 1908, 6.—Lafon & Martin, Rev. vet., 1908, 69.—Lignières, Rec., 1903, 725.—V. Ratz, A. f. Tk., 1893, XIX, 433.—Sequens, Vet., 1893, 23.—Thiroux or Teppaz, Rev. gén., 1907, IX, 345.

#### (b) Dochmiasis of Cattle.

**Occurrence.** Disease of cattle due to *dochmius* infection is generally rare; it has, however, been seen by Dawson in Southern Texas, and generally in Florida, where it is known under the name of "salt sick." Daschanek saw an enzootic of dochmiasis in Bohemia, among calves 5 to 12 months old.

**Etiology.** The disease is caused by *Dochimus radiatus* (*Uncinaria radiata*, *Anchylostomum radiatum*), a worm 7.5 cms. long (according to Duschanek only 1.5-2.8 cm. long); its cephalic end is curved like a hook. The ova contained in the feces of old, infected animals soon develop an embryo, under favorable conditions after 24 hours; in water or moist soil the latter undergoes several castings and is able to develop further in the intestinal canal of a proper host after 4 to 6 weeks (Stiles).

**Natural infection** occurs on pastures, especially where there is much stagnant water, or in the barn. Duschanek saw an enzootic of the disease in a barn after the animals had been drinking from a small brook which had become quite low in consequence of prolonged dry season.

Predisposing causes are poor pastures, unfavorable weather, difficult parturition, early weaning of calves, digestive disturbances.

**Anatomical Changes.** Aside from the signs of profound anemia, hydremia and cachexia, one observes catarrhal inflam-

mation of the duodenum with hemorrhagic spots in the mucosa, many *Dochmii* in this portion of the intestinal tract, partly adherent to the mucosa, partly free in the lumen, and also present to a lesser extent in the jejunum. The intestinal contents may be hemorrhagic.

**Symptoms.** A morbid desire to lick often is the first marked symptom of the disease; sometimes, however, this symptom appears later in its course and progressive emaciation becomes noticeable before anything else. Colicky symptoms appear from time to time, which are followed by diarrhea, lasting for several days. Diarrhea is also noticed during the whole course of the affection, independently of colics, alternating with constipation and bloating. Edema of the larynx, of the extremities, and collections of fluid in the serous cavities come on, the animals become feeble, their gait is staggering. The cachectic animals finally cannot get up any more and they die in coma.

The course and duration of the disease vary very much according to the number of *Dochmii* present in the intestinal tract.

**Diagnosis.** A correct diagnosis depends upon finding characteristic ova of *Dochmii* in the feces on microscopical examination (see page 483). The affection must also be differentiated from gastro-intestinal strongylosis by a microscopical examination of the feces; ova of *strongylus* (see page 458) are much larger than those of *Dochmius radiatus*, and they are usually unsegmented. Enteritis paratuberculosis is distinguished from dochmiasis by continuous diarrhea with water stools, and often by the presence of acid-fast bacilli, which may be found in great numbers in scrapings from the anterior portion of the rectum.

**Treatment and Prophylaxis.** Aside from intense feeding, the vermifuges that are recommended for gastric strongylosis and ascariasis (see pages 458 and 479) should be employed.

As prophylactic means are recommended, change of pasture (infected pasture may be utilized for other species of animals), clean drinking water, if possible running water, well or spring water, burning or burying of the manure, or at least frequent sprinkling of the manure with lime water, cleaning and drying out of the barn.

**Literature.** Dawson, D. v. W., 1907, 243 (Review).—Duschaneck, T. Z., 1909, 114.

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**Dochmii in Other Animals.** *Dochmius cernuus* lives occasionally in the small intestines of goats and sheep, without having a detrimental effect. Ströse saw digestive disturbances caused in a hog by *Dochmius longemucronatus*. What Ráthonyi described as a dochmiasis in nine horses, was a strongylosis, as shown by v. Ratz. Schmaltz found *Uncinaria os papillatum* in two elephants with hemorrhagic enteritis, anemia and hydremia.

(d) **Trichotrachelidae.**

*Trichina spiralis* (see trichinosis) is the only one of the fili-form *Trichotrachelidae* which is of pathologic significance.

**Trichocephali** are comparatively frequent, but apparently harmless inhabitants of the intestinal tract of ruminants, hogs and dogs. Their anterior end is very fine and filamentous, the caudal end is much thicker, cylindrical, straight in the female, rolled up in the male. The small ova, (fig. 59), are lemon shaped with stopper-like formations at either end.

The following species should be mentioned:

*Trichocephalus affinis*, 6 to 8 cm. long; found in the cecum and small intestine of ruminants, also in the intestinal tract of hogs (Meyer).

*Trichocephalus crenatus*, 4 to 5 cm. long; occurs in the large intestine of the hog and occasionally causes intestinal catarrh.

*Trichocephalus depressiusculus*, 4 to 7 cm. long; found in the cecum of dogs, particularly of those that suffer from dochmiasis. If present in large numbers it appears to cause catarrh of the cecum, occasionally also invagination (Mégnin), at other times it may be the cause of hemorrhagic inflammation of the colon (Miller).



Fig. 59. Ovum of *Trichocephalus affinis*.

Several species of **Trichosomata** with a thin anterior portion of the body, gradually becoming thicker towards the caudal end, are found in the intestinal tract of fowl (*Tr. longicolle*, *annulatum*, *collare*, *tenuissimum*, and *brevicolle*); one species is found in sheep. *Tr. tenuissimum* occasionally occurs in the intestinal tract of pigeons, sometimes in very excessive numbers, and then causes profound enteritis with secondary anemia with numerous fatal cases. Freese observed an epizootic of severe intestinal catarrh due to *Tr. retusum*; the disease was generally fatal in 3 to 10 days in young chicks, and in adult chickens, in 2 to 4 weeks. During the initial stage of the affection, oil of anise given in 5 to 10 drop doses to a tablespoonful of olive oil, proved very efficient.

**Literature.** Freese, D. t. W., 1908, 715.—Klee, Vet. Jhb., 1906, 352.—Meyers, Z. f. Flhyg., 1905, XIV, 157.—Miller, Am. V. Rev., 1904, 722.—Willett, O. M., 1906, 366.

(e) **Whipworms in the Intestine. Oxyuriasis.**

**Etiology.** The following *Oxyuris* occur in domestic animals:

1. *Oxyuris equi* (*B. curvalis*), the female alone is found almost exclusively in the intestine of the horse, 4 to 5 cm. long, thickened and curved in front, pointed behind, mouth surrounded by three lips; the male is difficult to find, 9 to 12 mm. long; oval ova (fig. 60), 88 to 95  $\mu$  by 41 to 44  $\mu$ , provided on one pole with a lid-like formation. The females sometimes are up to a size of 15 cm. and their shape is then changed so that the posterior more slender portion is 3 or 4 times as long as the anterior portion. Nitsch, Friedberger, and more recently Jerke, have claimed that this is another species which has received the name of *Oxyuris mastigodes*.

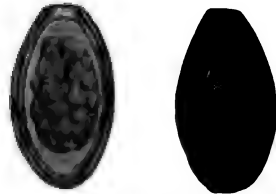


Fig. 60. Ova of *Oxyuris equi*.

2. *Oxyuris vermicularis*, 2 to 3 mm. long, posterior end rolled up, female 9 to 10 mm. and straight. Ova oval, flat on one end, convex at the other. Frequent found in the rectum of man; according to Zürn also in dogs.



3. *Oxyuris ambigua* occurs in the cecum of rabbits and there produces violent inflammation.

The development of *Oxyuris* in horses is, according to Jerke, as follows: the female deposits its ova at the anus of the host, while keeping itself there, fastened with its tail. The ova adhere to the skin around the anus and of the perineum, with a sticky substance, and they develop into embryos within 1 to 3 days. They then dry and drop to the floor, with detached scales of epidermis. They get out to the pastures with the manure, and may infect the feed and straw; these embryos remain alive quite long. (Roger claims that ova of *Oxyuris* are disseminated by flies.) After ingestion into the stomach of a host, the plug closing the ovum becomes dissolved, the embryo is set free and transported towards the large intestine, where it reaches maturity. Four months elapse between the time of infection and the appearance of mature females of *Oxyuris mastigotes*; of *Oxyuris currula*, about three months. Without free oxygen the embryos cannot develop in the ovum, hence this does not occur in the intestine of the horse.

Roger claims that *Filuria irritans* (see dermatitis granulosa) is nothing else but the larval stage of *Oxyuris*.

**Symptoms.** Female oxyuris, attached near the opening of the anus, sometimes causes an acute catarrh, or more frequently an intense itching in the region of the anus, as a consequence of such irritation the animals rub their hair and the neighboring parts on hard objects and sometimes produce eczematous inflammation, which may spread along the perineum and to the thighs. One finds then worms in the crusts, around the anus and also on the outer surface of the feces, and on rectal exploration on the arm of the examiner after withdrawal of the former; ova are found on microscopic examination (Pflug, Roger).

Friedberger reported the case of a mare which had exhibited signs of helminthiasis for two years, and which had voided 10 to 15 *Oxyuris mastigodes* daily, for 9 days; Illy observed severe colic which was cured permanently after the evacuation of worms. In a case of eczema of the tail with subsequent phlegmonous abscess, an *Oxyuris mastigodes* was found anchored into the perineal muscles by Hahn.

**Treatment.** The best treatment consists in repeated rectal injections of vinegar and soap water, possibly also solution of corrosive sublimate (1:2000). The internal administration of vermifuges is necessary only exceptionally in very obstinate cases (in Illy's case, arecanut 100 gm., followed in 4 hours by aloe 45.0 gm., had a good effect).

**Literature.** Friedberger, Münch. Jhb., 1882-83, 81.—Illy, Vet., 1893, 475.—Jerke, Zur Kenntn. d. Oxyuren d. Pferdes, Diss., Jena, 1901 (Lit.).—Pflug, Ö. Rev., 1881, 82.—Roger, Bull., 1905, 479.

(f) **Palisade Worms in the Intestines. Strongylosis intestinalis.**

(a) **Palisade Worms in Horses. Sclerostomiasis equorum.**

**Etiology.** *Sclerostoma* occur in the intestine of the horse. The description here given is based upon that furnished in 1900

by Looss and the careful investigations of Sticker, published somewhat later (1901).

The following three species belong to the genus **Sclerostomum**:

1. **Sclerostomum equinum**, Müller (*Strongylus armatus*, Rudolph; *Str. neglectus* Poepel, *Scl. quadridentatum*, Sticker). The male is on an average 35 mm. long, 1.25 mm. thick; the female 45 to 47 mm. long and 2.25 mm. thick. Mouth capsule almost regularly long-ellipsoid, with 4 high narrow teeth below them, the dorsal ones densely crowded, the ventral ones separated. The bursa at the tail end of the male wider than long, the median flap developed to a lesser degree. The genital pore of the female about 14 mm. in front of the caudal end. Ova oval, thin shelled, 65 to 80  $\mu$  long by 45 to 50  $\mu$  wide. Larvæ 0.58 to 0.88 mm. long, similar to those of *Scl. edentatum* only shorter; its anterior end somewhat conically pointed (Albrecht).

2. **Sclerostomum vulgare**, Looss (*Strong. arm.*, Rudolph; *Scl. bidentatum*, Sticker) claimed to be a young form of the preceding one by Rudolph; however, really a new species, the larvæ of which are concerned in the production of so-called worm aneurysm (see page 393). Male at the utmost 15 or 16 mm. long and fairly uniformly 0.7 mm. thick; female 23 or 24 mm. long and 1 mm. thick, slender towards the tail end, mouth capsule like a shallow goblet. Two teeth at the lower end of the dorsal depression of the mouth capsule; these protrude as ear-like projections. Bursa of the male with three flaps, the median one, larger than that of the preceding species, is overlapped by the lateral flaps. The female genital pore 8 mm. anterior to the caudal end. Ova 70 to 80  $\mu$  long and 43 to 52  $\mu$  wide. The anterior end of the larvæ (0.7 mm. long) is somewhat rounded off, the posterior portion gradually diminished into a filiform tail end, which is about one-half shorter than the body. Around the gut 32 larger cells are usually arranged in double rows in mosaic form; the sexual apparatus is situated on a level with the 13th intestinal cell (Albrecht).

3. **Sclerostomum edentatum**, Looss (*Scl. edentatum*, Sticker). Length of male 23 to 25 mm., greatest thickness about 1.5 mm.; length of female 33 to 36 mm., thickness about 2 mm. Head globular, distinct from body; mouth capsule goblet-like, teeth-like formations totally absent; however, at the place where the ventral teeth are situated in *Scl. equinum*, there are some small nodular elevations. Bursa of the male very similar to that of *Scl. equinum*. Genital pore of female 9 to 10 mm. anterior to the caudal end. Ova (Fig. 61) 65 to 88  $\mu$  long and 45 to 50  $\mu$  wide. Larvæ 0.55 to 0.88 mm. long, body almost one-third longer than the gradually tapering caudal end; marking of the intestinal cells indistinct, thickness of the body less than that of larvæ of *Scl. vulgare* (Albrecht).



Fig. 61. Ovum of *Sclerostomum edentatum*.

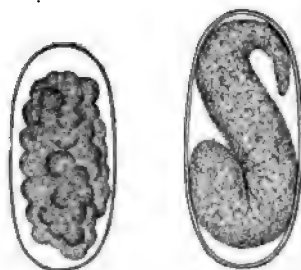


Fig. 62. Ova of *Cyathostomum tetracanthum*, on the right side ovum with embryo from horse manure preserved one day.

Of the genus **Cyathostomum**, the most important species is the one generally designated as *sclerostomum tetracanthum* or *strong. tetracanthus*:

**Cyathostomum tetracanthum** (*Strongylus tetracanthus* Mehlis, *Sclerostomum tetracanthum*), white worm; male about 9 mm., female 10 to 12 mm. long. Mouth capsule fairly shallow, narrowed in the middle, fine triangular leaflets, arranged in a radial manner and looking from above like a wreath of fine bristles (called by Looss internal leaf wreath) spring from the narrowed place of the mouth capsule. In front of the anterior margin of the mouth capsule there is an outward projecting



wall formed by the skin, this wall is likewise provided with radially arranged leaflets (external wreath of leaves). Sexual pore at the posterior end, directly in front of the anus; ova (fig. 62) oval 90 to 110  $\mu$  long, 40 to 50  $\mu$  wide. The caudal end of the larvæ (0.46 to 0.80 mm.) is about one-third longer than the body proper, anterior end of the latter somewhat more pointed, the union of the body proper and the tail end well marked; the intestinal tract only shows 8 or 9 larger alternating cells, on a level with the fifth of these cells the sexual apparatus is situated (Albrecht).

As the representative of a third genus Looss distinguishes *Triodontus* which has an almost globular small but thick-walled mouth capsule, which possesses no real teeth, but the chitinous lining of the three-cornered lumen forms three projecting tooth-like formations. The genital pore of the female is situated near the caudal end. Two species belong to this genus: *Triodontus minor* and *Triodontus serratus*. They do not, however, appear to occur in Europe.

According to Leuckart, Cobbold and A. Albrecht, the **development** of *Sclerostomum* and of *Cyathostomum tetracanthum* is as follows: In the ova, which are voided with the feces of the host, embryos develop in the outside world, under favorable conditions of temperature, in 3 to 4 days, later in winter, which after hatching, become larvæ 0.4 to 0.8 mm. long, round with filiform, caudal ends. In water or in dry dust, the larvæ can live only a few days, but they may remain alive for months in moist or at least not too dry horse manure. After 15 to 20 days, or at a temperature of 30° C., shedding takes place in a few days; the detached cuticle is completely shed, however, only after several months. With this shedding, the resistance of the larvæ is much increased, and it may now not merely survive in horse manure for several months, but also in pure water for 6 to 8 months, and it is not even killed at freezing temperature (0° C). The hatched embryos probably go through three or four moults, although Albrecht observed only one shedding. After slipping out of the shell, the larvæ lose their whiplike tail end and they are frequently designated as rhabditis forms.

**The ingestion of larvae** occurs with contaminated drinking water, with feed contaminated with manure or on infected pastures. The infection is not confined to certain seasons as was assumed by Sticker.

The views of authors differ as to the further fate of the ingested larvæ. According to the views of Leuckart, Railliet, Sticker, Glage, the larvæ bore into the intestinal wall immediately after ingestion, get into the general circulation and are transported to various organs. Larvæ of *Scl. bidentatum* then settle in certain arteries (see page 393) from where they are washed away after several weeks or months (Glage); they then remain for several weeks in small nodules of the intestines (especially in the cecum, Sticker), then they get again into the intestinal lumen where they now copulate. Larvæ of *Scl. edentatum* from the abdominal organs get into the intestine by active wandering (Glage, Schlegel) and they produce mechanical lesions in these organs. According to another view (Colin, Willach, Olt), larvæ found in the blood vessels and other organs are only looked upon as individuals that have gone astray and that can no more contribute to the preservation of the species.

**Anatomical Changes.** No changes are noticeable in the mucosa of the large intestine in the presence of mature individuals of *Sclerostomum quadridentatum* and *Sclerostoma bidentatum*, but the larvæ of *Scl. bidentatum* produce thrombosis, particularly in the mesenteric arteries (see page 393). *Cyathostomum tetracanthum* may cause hemorrhagic inflammation of the large intestine. In acute cases of sclerostomiasis caused by the larvæ of *Sclerostoma edentatum* one finds a purely serous,



sometimes a little cloudy, exudate with flocculi of fibrin. The subperitoneal areolar tissue is in a condition of edematous infiltration, amber in color, and contains hematomata of the size of a walnut up to that of an egg, and hemorrhagic spots, particularly in the parietal portion of the mesentery, the intestinal serosa, and also often in the perineal fat. The hemorrhagic spots and foci contain each one mature worm, which shines through like a varicose vein if situated superficially. Exceptionally, there may be a fatal peritonitis after perforation of a nodule by the worm it contained. The intestinal mucosa may show diffuse or circumscribed hemorrhages. The submucosa contains worm nodules, the size of a millet seed up to that of an almond, with reddish-yellow or purulent contents, in which the larvæ are situated, or they may already have migrated into the intestinal lumen. The anatomical findings in the most profound cases include the signs of anemia, emaciation or cachexia.

**Symptoms.** Mature worms of *Sclerostoma quadridentatum* and of *Scl. bidentatum* do not cause any disturbance of health, as has already been stated, while the larvæ of *Scl. bidentatum* cause thrombotic closure of various blood vessels. Continued intestinal hemorrhages, with emaciation, occur in the presence of *Cyathostomum tetracanthum*; in other cases only short attacks of diarrhea with colic. Numerous cases of disease have also been observed (Schwarzmeier, Penberthy, Williams, Bochsberg) especially in foals kept on wet pastures. Some of the affected animals, particularly foals, succumb to the disease.

Of much greater importance is that form of sclerostomiasis (sclerostomiasis enzootica, Schlegel) that is caused by the larvæ of *Sclerostomum edentatum* and almost exclusively occurs in foals. It often manifests itself only in a progressive anemia and emaciation, extending over months and even years; otherwise the body temperature and the pulse are normal (Glage); in some cases, however, particularly after exercise, colicky symptoms are observable (Kläber).

In other cases the clinical symptoms are more pronounced and are similar to those of a peritonitis with colic (Schlegel). The temperature rises rapidly to 40-41.9° C., sometimes only to 38.5-39.6° C.; the pulse up to 78-80-100 per minute; it becomes weak and finally can no longer be felt. Respiration is rapid and accompanied by groaning. There is great prostration, lack of appetite, yellowish or brownish-red discoloration of the mucosa. Occasionally there are attacks of a mild colic, urination is exceptionally frequent and the urine shows a red discoloration. The symptoms gradually become more intense and finally profound anemia and cachexia develop. Most patients die after a few days (acute form); in others the disease lasts up to seven weeks. Some animals, however, are not very seriously sick and recover, while those affected profoundly all die. Isolated hemorrhagic spots with larvæ of *sclerostoma* are frequently found in

horses that are otherwise healthy, upon being killed in the slaughtering house.

**Diagnosis.** The presence of mature *Sclerostoma* in horses may be recognized frequently on rectal examination when some of the worms are brought out on the arm of the examiner. As a rule, however, the diagnosis is made only upon finding ova by microscopic examination of the feces.

The methods of Adelmann and A. Albrecht permit the differentiation of the larvæ of the various species of *Sclerostoma*. A ball of feces is placed into a glass vessel and this is wrapped in black paper so as to be protected against the light, covered to prevent desiccation and left in the neighborhood of a stove or in another warm place. After two days the feces then contain lively motile embryos (Adelmann). According to Albrecht's method the ball of feces is preserved for 8 to 14 days or in summer for 5 to 8 days, and protected during this time from desiccation. Clean water or physiologic salt solution is then poured over the ball, enough of the former so that it is fully soaked and there is some excess on the bottom of the vessel. After a few hours one may recognize, if the light is favorable, minute worms or little conglomerated masses of them in the water at the bottom of the vessel with the naked eye. Sometimes the worms can be recognized only after the water has been carefully poured off or centrifuged. With the aid of the microscope the larvæ of the various species can be differentiated according to the characteristics described by Albrecht.

Since *Sclerostoma* may be present in the intestine of the horse without giving rise to any disturbance in health, it is necessary, in the presence of symptoms of disease in which *Sclerostoma* are found, to make a careful examination of the animals and to consider all conditions of environment before the morbid condition can be ascribed to sclerostomiasis. Bloody diarrhea in young horses in summer is always suspicious of sclerostomiasis.

The differential diagnosis must consider the so-called wasting of foals (Dieckerhoff) (see page 342), especially also infectious anemia (see Vol. I); the latter is very similar in its clinical and pathologic characteristics to sclerostomiasis as described by Schlegel.

**Treatment.** To combat intestinal sclerostoma one may make use of the same means which are recommended for ascariasis (see page 479). Albrecht recommends particularly oil of turpentine (80 gm. with 500 gm. of castor oil); Dorn praises intravenous injections of atoxyl (3 gm. in 100 cc. warmed to 37° C.) Boehberg also recommends atoxyl (he gives it in doses of equal amount, or gradually increasing from 0.2-0.5 or 1-1.5 gm. in 1 per cent salt solution, administered subcutaneously or intravenously). Atoxyl or sodium arsenicosum (0.5-2.0 gm.) in increasing doses internally might also be used against the larvæ of *Sclerostoma*. The animals should have the benefit of intense feeding.

Prophylaxis is the same as that recommended against thrombosis of the mesenteric arteries (see page 410).

**Literature.** Adelmann, *Das Aneurysma verm. equi etc.* Diss., Giessen, 1908 (Lit.).—Albrecht, *Z. f. Vk.*, 1909, 161.—Boehberg, *Z. f. Vk.*, 1909, 271.—Dorn, *M.*

t. W., 1909, 109 (Ref.).—Glage, Z. f. Infkr., 1906, 1, 341 (Lit.).—Kläber, B. t. W., 1891, 223.—Looss, Cbl. f. Bakt., 1900, XXVII, 150 (Lit.).—Schlegel, B. t. W., 1907, 49.—Sticker, A. f. Tk., 1901, XXVII, 187 (Lit.); D. t. W., 1901, 253.

(b) *Oesophagostoma* in the Intestines. *Oesophagostomiasis intestinalis*.

(*Oesophagostomiasis nodularis* [MAROTEL]; *Maladie nodulaire, Helminthiase nodulaire ou larvaire, Folliculite caséuse, Dochmiase larvaire* French].)

**Occurrence.** Diseases caused by the larvæ of *Oesophagostomum* occur in all parts of the world, but preferably in Europe and America, mostly sporadic but also enzootically, and then with high mortality. Cattle and sheep are affected most commonly, more rarely goats and pigs. *Oesophagostomas* are also occasionally found in the intestines of deer, monkeys and even in man.

**Etiology.** *Oesophagostomas* have no mouth proper because the opening representing the mouth leads directly into the esophagus. Mature individuals of *O. radiatum* have a length of about 15-20 mm. and a ring-shaped mouth capsule, a cuticular swelling at the neck, a ventral furrow, and a bursa at the posterior end (Marotel).

The following *oesophagostomas* occur in domestic animals:

1. *Oesophagostomum radiatum* in the intestines of cattle, there first found by Drechsler (1876), later also by Saake, Giles, Jansen, Ströse, v. Rätz, Scheben and others.

According to Marotel the parasite is identical with *O. inflatum* Railliet *Anchylostomum bovis* Ströse, and *Anchylostomum radiatum* Scheben. Marotel succeeded in finding the mature form of the larvæ living in the intestinal wall, and in showing that it properly belongs to the genus *Oesophagostomum*.

2. *Oesophagostomum venulosum*. This worm occurs in European sheep and goats.

3. *Oesophagostomum columbianum* lives in the intestines of sheep in America.

4. *Oesophagostomum dentatum* occurs in the intestines of swine and is identical with *Strongylus follicularis* Olt.

The development of *Oesophagostomum* takes, according to Marotel, place, as follows: From the ova voided with the feces of the animal host, embryos are at once hatched if heat and moisture are sufficient. The further development of the latter in the outside world is, however, not yet known. If the larvæ are taken up during the months of August and September, they bore at once into the intestinal wall, and remain for 6 to 7 months in the submucous connective tissue, where they successively develop, first, into strongyliform, then anchylostomiform, finally *oesophagostomiform* larvæ. During March and April, they wander into the intestinal lumen, and here they attain sexual maturity during May and June, their full length only in July and August, then they copulate, and the females deposit their eggs. The maturation of *Oesophagostomum dentatum*, however, occurs within the intestinal worm nodules (Seiler); the same is also the case in the *Oesophagostomum* living in the intestines of man.



**Natural infection** takes place, according to Marmotel, on wet, marshy pastures during the months of August and September; but it probably takes place also during sojourn in the barn if the latter offers conditions favorable to the development of embryos from the deposited ova.

The pathologic significance of the larvæ of *Oesophagostomum* living in the intestinal wall is not solely confined to a detrimental effect upon the function of the intestine by numerous worm nodules, but also in the absorption of numerous pathologic bacteria for which these nodules form a portal of entrance; this may become the cause of secondary infection.

**Anatomical Changes.** In the small intestine and cecum of cattle, and in the large intestine of other animals, tubercular nodules (roundworm nodules) are found often isolated; frequently also in great numbers, in hundreds and thousands. (Drechsler once counted 430; Marotel saw in one case 4,000 in the small intestine and 1,000 in the large intestine.) The nodules vary from the size of a pinhead to that of a pea, and they may occasionally be as large as a hazelnut; the smallest ones have a blackish color, the medium-sized nodules are blackish to grayish-white, the larger ones are always grayish-white. The otherwise unchanged intestinal mucosa shows half-spherical projections; it appears somewhat thinner at this place and in hogs it is also ulcerated. The nodules are mainly composed of a thick connective tissue capsule which may contain a small amount of caseous, and in the larger nodules purulent, matter; the latter may have become calcareous. The younger nodules contain larvæ of *Oesophagostomum* measuring 3-4-7 mm.

**Symptoms.** A few nodules are frequently found, in slaughter houses, in the intestines of animals which did not show any symptoms of disease during life. Morbid symptoms are only observed in intense invasion and they consist in untractable profuse diarrhea, progressive emaciation, anemia and cachexia. The disease then usually terminates fatally in coma which comes on after two to three months. When the disease appears as an enzootic one-half of affected animals may die.

**Diagnosis.** The occurrence of the above mentioned symptoms following sojourn on wet, marshy pastures may suggest oesophagostomiasis nodularis, but a reliable diagnosis can only be made on the basis of some postmortem examinations, since the ova of *Oesophagostomum* are not yet known; the differential diagnosis has to consider gastro-intestinal strongylosis (see page 457) and enteritis paratuberculosa (see Vol. I).

**Treatment.** This must be confined to a forced nutrition of the patients. Prophylactic measures are those recommended for gastric strongylosis (see page 458).

**Literature.** Drechsler, D. Z. f. Tm., 1876, II, 355.—Marotel, Journ. Vét., 1908, 522 (Lit.).—v. Rätz, B. t. W., 1891, 223.—Scheber, Fortschr. d. Vet.-Hyg., 1905, 97.—Seiler, D. t. W., 1902, 345 (Lit.).

**Other Filiform Worms in the Intestines.** *Strongylus ventricosus* occurs sometimes in the small intestines of cattle and sheep without causing disturbance of health. *Strongylus filicolis* occurring in the duodenum of sheep, may cause enteritis and intestinal hemorrhages. *Str. nodularis* occurring in the duodenum of geese and *Str. tenuis* parasitic in the cecum of geese, chicken and ducks, is without any pathologic significance.

*Sclerostomum hypostomum* is frequently found in the large intestine of sheep and of goats (see fig. 63); it occasionally produces intestinal hemorrhages.

*Filaria papillosa* found frequently in the abdominal cavity or under the peritoneum of horses, according to Rudolphi, occurs occasionally in the intestines of this animal. It has, however, no influence upon the health of the host. *Rhabdonema longum*, a small worm only 6 mm. long, is a rare inhabitant of the intestine of the sheep; similar to it is *Rhabdonema suis* found in the intestines of swine.

*Anguillula stercoralis* is, according to Zürn, found in Cochin China chickens and causes diarrhea.



Fig. 63. Ovum of *Sclerostomum hypostomum*.

#### (g) *Echinorrhynchus Gigas* of Swine.

**Etiology.** The *Echinorrhynchus gigas* is a thick, cylindrical, white worm, thicker at the anterior end; its external surface appears annulated; an intestinal canal is not present. The protrusible, rounded cephalic end is armed with five or seven rows of strong hooks; male 6-9 cm., female 20-35 cm. long; ova (Fig. 64) oval; the embryo is visible within a triple envelope; the head of the embryo is provided with hooklets.

The following may be said about the development of *Echinorrhynchus gigas*: According to Schneider, the ova are taken up with the larvæ of the cockchafer (May-bug, *Melontha vulgaris*). The free embryos perforate the intestines of these Arthropodes, become encapsulated in their abdominal cavity and remain alive even in the adult May-bugs. If hogs feed on May-bugs or their larvæ, the capsule of the parasitic larvæ becomes dissolved in the intestinal canal, the larvæ then bores into the intestinal wall of the host. *Echinorrhynchus* larvæ can probably go through their development also in other animals, aside from the May-bug. This seems to be proven by the fact that the worm also occurs in America where May-bugs do not exist. According to Lespes, ech. larvæ occur in certain snails; according to Kaiser and Blanchard, in *Cetonia aurata*; according to Stiles, in *Lachnosterna*.



Fig. 64. Ovum of *Echinorrhynchus gigas*.

*Echinorrhynchus* is found with marked frequency in some years and then causes great loss among young pigs. It appears

as if this periodic frequency stands in relation to the three to four years period of development of May-bugs.

**Anatomical Changes.** Grayish nodules or yellowish purulent points surrounded by a red halo are seen shining through the serosa of the intestines (Kocourek). The worms bore into the mucosa with their anterior ends; the neighborhood of the mucosa where the parasites are located is intensely reddened and also shows catarrhal swelling. Sometimes the parasites have perforated the intestinal wall and have produced a purulent peritonitis. Sometimes the mucosa shows cicatricial formations.

**Symptoms.** Digestive disturbances occur proportionately to the number and to the stage of development of the worms; the animals become emaciated and enfeebled. Since the parasites cause pain, the animals grunt continually; they are restless, bury into the ground or straw, snap towards the abdomen or towards other animals. Occasionally, particularly in young pigs, one sees more or less intense twitching of the muscles or epileptiform convulsions, generally followed by a fatal termination.

The cause of these symptoms is revealed, by a microscopic examination of the feces, by the discovery of the characteristic ova; frequently, however, such an examination is neglected and the true cause of the disease is only found on postmortem examination.

**Treatment and Prophylaxis.** These worms can be expelled only with difficulty. One may try drugs that are effective against ascaris, especially santonin (2.5 gm.), rhizoma filicis maris (20-50.0 gm., or of the extract 2.5.0 gm.), kamala (2-4.0 gm.); also, according to the recommendation of Kocourek, oil of turpentine (in tablespoonful doses, followed by laxatives, see page 480).

The destruction of insects and other animals, which may contain the larval stage of the worm, is indicated as a prophylactic measure, also the avoidance of probably infected parts of land.

**Literature.** Kocourek, Trzt., 1887, 256.—Stiles, J. of comp. Path., 1891, 657.—Weinberg & Romanovitch, A. P., 1907, 960.

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**Echinorhynchus in Fowl.** *E. polymorphus* is found in the intestines of ducks, more rarely in the intestines of geese and swans; in ducks also *E. filicolis* and *E. sphaerocephalus*. The first sometimes causes epizootics (Klee, Vet. Jhb. 1906, 352).



### 30. Coccidia in the Intestines. *Coccidiosis intestinalis*.

#### (a) Red Dysentery of Cattle. *Dysenteria coccidiosa bovom*.

(*Kokzdienruhr* [German]; *Entérite hémorrhagique, Flux de sang* [French].)

Red dysentery of cattle is a disease preferably of young animals, generally occurring as an enzootic affection, and due to *Coccidium Zürn*; these are generally found in the large intestines, preferably in the rectum. The disease manifests itself particularly by hemorrhagic defecation.

**Historical.** Zürn was the first (1878) who found coccidia in the inflamed intestines of a calf; it was, however, only in 1892, that Zschokke pointed out the etiologic relation between coccidia in the intestines and red dysentery. Hess (1892) and Guillebeau (1893) confirmed the findings of Zschokke, and Guillebeau made culture and inoculation experiments. Others who have studied this disease were Degoix (1904), Züblin (1908), also Bugge, Warringsholz & Sieg (1909).

**Occurrence.** The disease occurs almost exclusively during the warm season between the months of June and September and especially in wet years; it may, however, be observed here and there later in the fall and even in winter. Commonly (95 to 100 per cent) animals become infected on the pasture and preferably in mountainous parts, more rarely in lowlands.

The disease has been observed particularly in the mountainous cantons of Switzerland, in certain parts of France, but it has also occurred in Denmark (Poulsen), in North Germany, and has been observed in Hungary by Makoldy.

**Etiology.** *Coccidium Zürn* (*Eimeria*) is, according to Martin and Züblin, a distinct species; according to Zschokke, Hess and Guillebeau, it is, however, probably identical with *Coccidium cuniculi*. Coccidia are found in the feces of sick animals as round or oval formations (oocysts) with a long diameter of 10-20  $\mu$  according to Zschokke, 18-25  $\mu$  according to Degoix, and 15-25  $\mu$  according to Züblin; the width varies between 10-20  $\mu$ . The highly refractive protoplasm is finely or coarsely granular or globularly contracted (Fig. 65; it shows a light spot in the center (nucleus). There are also seen hyaline folded membranes; these are probably dead coccidia. The membrane of unstained cells appears shining and peculiarly greenish violet in color.



Fig. 65. Intestinalcoccidia. (According to Degoix.)

**The development of coccidia** is as follows: In the oocysts voided with the feces there appear four globular sporoblasts, if the conditions of moisture, heat and access to free oxygen are favorable, within 2 to 6 days; these assume a spindle shape and become sporocysts; in these two crescents or sickle-shaped sporozoites are developed. These changes may occur in 4 or 5, or only in 14 or 15 days, accord-

ing to environment. The oocysts are very resistant and can remain alive after putrefactive processes lasting for three months (Degoix). After oocysts have been ingested their membrane is dissolved by the digestive juices, particularly by the pancreatic juice (according to Züblin in 6 to 8 hours), they are set free and then form the lively motile sporozoites which penetrate into the intestinal epithelia and assume a roundish form, being then found between the cell margin and the nucleus. They enlarge gradually and become schizontes, which multiply asexually (schizogony) by forming numerous spindle-shaped merozoites. After the dissolution of the infected epithelial cells these get into the intestinal lumen and invade new epithelial cells; the process of schizogony is repeated several times; some merozoites situated in epithelial cells then change into macrogametes and microgametes. After the latter have become free they penetrate into macrogametes with active motility, fertilize these and form again an oocyst (sporogony). Whether asexual multiplication may also occur outside of an animal host is not known, but Züblin believes that it is possible.

The **infection** is spread by the water of pools and marshes, also probably by feed which has come in contact with infected water. (Zschokke claims that the coccidia first get into snails and that the latter infect the water or feed.) The possibility of infection in the barn is suggested by the fact that isolated cases also occur in winter in infected regions, also by the occurrence of coccidiosis among other species of animals which are not pastured, and by an observation of Zürn that calves became infected which had been confined permanently to the barn. Damp and dirty barns undoubtedly are particularly dangerous.

Three young cattle, experimentally fed on material containing sporozoites fell sick after three weeks with the symptoms of acute intestinal catarrh, with diarrhea and few coccidia in the feces, but without any blood. These symptoms lasted three days (Guillebeau). The disease cannot be transferred by non-sporozoitic oocysts. Züblin did not succeed in infecting rabbits with coccidia from cattle that were suffering from red dysentery.

The susceptibility is greatest in youth, up to the second year of life, but older cattle likewise contract the disease, although more rarely. The great rarity of the disease among calves less than six months old may be explained by the fact that these animals are usually not pastured. Isolated cases have, however, been observed among calves three to six weeks old (Zürn, Bugge, Waringsholz & Sieg).

**Pathogenesis.** The sporozoites escaped from the ingested oocytes, wander with the intestinal contents toward the anus and penetrate into the epithelial cells of the crypts of Lieberkühn of the large intestine, particularly into those of the rectum. They multiply rapidly by schizogony and the epithelial cells of the surrounding glands and those of the mucosa in general are invaded by autoinfection; in this manner an increasing area of the mucosa becomes affected; the usual localization of the changes in the large intestine is attributed by Züblin to a comparatively slow solution of the oocyte membrane and to a rapid passage of the contents of the small intestine into the large intestine. It is, however, remarkable that the small intestine is also profoundly affected in young calves and that



the small intestine of other species of animals is most profoundly affected.

After the epithelial cells have become invaded they are shed and this produces a denudation of the glands and of the mucosa, and the congested capillaries protrude free towards the intestinal lumen. This explains the occurrence of intestinal hemorrhages. At the same time irritation by coccidia produces a small round cell infiltration of the mucosa and of the layers below it. Since the mucosa becomes denuded in spots and deprived of its protecting covering, an intense invasion of debilitated animals may lead to secondary infection by intestinal bacteria and this may produce severe enteritis and general infection.

**Anatomical Changes.** One usually finds the intestines contracted; in the large intestine and particularly in the rectum one finds thin fluid greenish or reddish-gray, to reddish-brown contents, sometimes containing distinct blood coagula or even ichorous masses. The mucosa of the rectum is usually affected most profoundly, the mucosa of the colon or cecum much less or not at all, although the latter sometimes shows profound changes. The mucosa shows strong corrugation, reddening, swelling and a mucoid, fibrinoid, yellowish or grayish deposit sometimes mixed with blood. In consequence of the loss of epithelial cells the surface of the mucosa appears rough in places with hemorrhagic spots or streaks. Epithelial losses and floating shreds of mucus or detached epithelial cells are seen in advanced cases, alternating with slate gray spots of the mucosa. Necrotic processes in the large intestine with catarrhal or hemorrhagic inflammation in the small intestine are frequently seen in consequence of a secondary infection. Numerous coccidia (Fig. 66) are seen in the intestinal contents and in detached shreds of the mucosa. The cadavers also exhibit the signs of profound anemia and cachexia.



Fig. 66. Position of intestinal coccidia in the glands of Lieberkühn. Low magnification. (According to Degoix.)

**Symptoms.** The peculiarities of the development of coccidia bring it about that the period of incubation of the disease is comparatively long, about three weeks; occasionally, however, symptoms may come on after six or eight days (Bugge, Warringsholz & Sieg). On the basis of his observations Degoix claims that the period of incubation is one or two months; he



does so because he saw the disease come on in cattle after they had been in the barn for one or two months. However, since infection may also occur in the barn, Degoix' claim does not seem to be well founded.

The disease begins immediately with diarrhea and after one or two days the feces show an admixture with blood and mucus; distinct blood coagula from the size of a walnut to that of a child's fist are also seen; the feces are fetid and sometimes have a cadaverous smell. As soon as hemorrhagic feces appear, there is at first slight, later on intense tenesmus, which may lead to prolapse of the rectum. In other cases normal feces are voided at first, followed by a small amount of coagulated blood, later on feces and blood are mixed and diarrhea supervenes.

In this stage mild cases soon pass into recovery, particularly in adult cattle; the blood disappears after two or three days, and after a few more days the diarrhea stops. The appetite, however, remains capricious for some time.

In grave cases, particularly in young animals, there is a rapid deterioration of the condition. Tenesmus becomes more severe, the feces become quite fluid, very fetid; they contain larger coagula and shreds of mucus; appetite and rumination are suppressed entirely. At the same time the emaciation progresses rapidly and the gait of the animals becomes staggering. The pulse rises gradually to 70-80 and later on even higher; the temperature rises to 40° C. and above. Later on the blood disappears from the feces, but they often contain lumps of mucus, like croupous membranes, and an ichorous thin fluid. The emaciation has now reached a high degree, the eyes are deeply sunken, the animals can no longer stand up and they die from complete prostration.

**Course and Prognosis.** The disease runs an acute course of five to ten days, but there are also hyperacute cases in which the animals die within twenty-four hours in convulsions. The appetite sometimes remains poor for a while after disappearance of the grave symptoms, so that the animals become much emaciated; anemia may then become so intense that the patients may die even after five months. In the mild, favorable cases, recurrent attacks are quite frequent, but these usually take a benign course. Very mild cases lead to the symptoms of simple acute intestinal catarrh.

As complications of the disease one can sometimes observe blackleg, convulsions and coma in pregnant animals, bronchitis and pneumonia.

The prognosis is the more unfavorable, the younger and the weaker the sick animals; the more rapidly the appetite decreases, the earlier rumination ceases. Two to four per cent of the sick animals usually succumb to the affection.

**Diagnosis.** The occurrence of the disease during the time of pasturing, the almost exclusive affection of young animals,

hemorrhagic diarrhea and the rapid emaciation suggest the diagnosis. The detection of the coccidia by microscopic examination of the feces makes the diagnosis absolute and excludes rectal hemorrhage of cattle (see page 325).

Coccidia are at first found in the diarrheic stools only sparingly; they appear, however, in large numbers in the shreds of mucus and they can, according to Züblin, be seen in the voided coagula. In the terminal stages of the disease the number of coccidia again decreases. In order to find the coccidia more easily Züblin recommends to stain microscopic cover glass preparations with Lugol's solution. Coccidia have not been found in the feces of healthy cattle even in infected herds.

**Treatment and Prophylaxis.** Pasturing should be interrupted and dry feeding instituted, if the disease appears in a herd. Disinfectants (creolin, lysol, resorcin, sodium thio-sulphuricum) have been used as long as the disease has been known; also astringents (tannic acid, argentum nitricum, ferum sulfuricum) in the form of rectal injections in weak solutions, or they may be given as a mucilage or with milk by the mouth. Züblin saw good results from rectal injections with 1 per cent tannic acid or 1 per cent alum. If the animals suffer from lack of appetite, they must be fed with milk and eggs to which claret might advantageously be added.

As a prophylactic measure the ingestion of dirty water from pools and marshes must be prevented; if the disease makes its appearance, the healthy animals should receive dry feeding, or they should at least be brought to dry pastures where they have access to pure water. The feces should be soaked with a 3 per cent watery solution of sulphuric acid; this kills the coccidia.

**Literature.** Bugge, Warringsholz & Sieg, D. t. W., 1909, 769 (Lit.).—Degoix, Rev. gén., 1904, III, 177.—Ducloux, *ibid.*, 1906, VII, 138.—Hess, Schw. A., 1892, XXXIV, 105.—Martin, Rev. vét., 1909, 280.—Zschokke, Schw. A., 1892, XXXIV, 1.—Züblin, Schw. A., 1908, L, 123 (Lit.).—Zürn, Vortr. f. Trzte., 1878, I, H, 2.

#### (b) Intestinal Coccidiosis of Sheep and Goats. *Coccidiosis ovum et caprarum.*

Coccidia have repeatedly been found in the intestinal tract of sheep, but the disease caused by them has only recently been studied by Moussu & Marotel. The latter has also studied coccidiosis of sheep. Suckling lambs as well as adult sheep are affected, the latter more frequently. The symptoms are those of progressive anemia, emaciation and diarrhea. Death occurs after several weeks from exhaustion; the feces do not become hemorrhagic, nor are any coccidia found in the discharges from the intestinal tract. The somewhat reddened mucosa of the small intestines shows whitish points, which are formed by masses of coccidia which have invaded the glands of Lieberkühn. This species of protozoa has been called *Coccidium Faurei*.

Intestinal coccidiosis in goats is caused by *Coccidium Arloingi* (Marotel); its exogenous development is like that of *C. cuniculi*. Pathologic changes are exclusively found in the small intestine of goats, their picture is on the whole similar to that found in coccidiosis of



sheep; a small round cell infiltration is seen in the neighborhood of the invaded tissue.

**Literature.** Marotel, Bull., 1906, 373.—Marotel & Moussu, 1901, 470.—Martin, Rev. vét., 1909, 341.

#### (c) Intestinal Coccidiosis in Carnivora.

Coccidia are rarely found in the intestinal villi of dogs and cats; they were originally held to be *Coccidium perforans*, but, according to Railliet, they are a special kind (*Coccidium bigeminum*, according to Martin *Diplospora bigemina*); apparently they do not produce any morbid symptoms.—Dages found coccidia in the epithelia of the intestinal mucosa which, according to Martin, appear to be identical with *Cocc. cuniculi*.

**Literature.** Martin, Rev. vét., 1909, 345.—Zürn, Vortr. f. Thieraerzte, 1878, II, H, 2 (Lit.).

#### (d) Intestinal Coccidiosis of Rabbits. *Coccidiosis intestinalis cuniculi*.

Intestinal coccidiosis of rabbits caused by *Coccidium perforans* (according to Martin *Eimeria cuniculi*) is observed either alone or in combination with hepatic or nasal coccidiosis, (see page 28), and under similar circumstances.

Anatomical changes are usually found in the small intestine, and they consist in an intense intestinal catarrh with accumulations of tenacious, somewhat reddened mucus on the swelled and moderately reddened mucosa. The mucosa also exhibits small whitish nodules, not larger than a lentil. These nodules represent enlarged glands of Lieberkühn and epithelial cells of villi invaded by coccidia.

The symptoms are either identical with those of hepatic coccidiosis (see there) with the development of a cachectic condition, or the affection runs the course of a violent enteritis with marantic diarrhea followed by death before emaciation has had time to occur.

The diagnosis is made either by the microscopic discovery of the coccidia which are present in large numbers in the feces, or by post-mortem examination. If hepatic coccidiosis alone is present, only few coccidia can be found in the feces.

The treatment is similar to that in acute intestinal catarrh (see page 330); it is, however, usually not successful. Systematic prophylaxis promises much better results. It is based upon the same principle as prophylaxis against hepatic coccidiosis (see there).

**Literature.** Martin, Rev. vét., 1909, 201.—Zürn, Vortr. f. Thieraerzte, 1878, I, H, 2 (Lit.).

#### (e) Intestinal Coccidiosis in Fowl. *Coccidiosis intestinalis avium*.

(*Weisse Ruhr des Geflügels* [German].)

**Occurrence.** Intestinal coccidiosis is observed among all species of domestic fowl, particularly among young animals, and in enzootic distribution during the warm season. McFadyean has seen coccidiosis among pheasants.



**Etiology.** *Coccidium tenellum* invades the intestines of fowl. Different species of coccidium described as *Coccidium Pfeifferi* (pigeons), *C. avium* (chicken and ducks), *C. truncatum* (geese and ducks), *C. oviforme* (pheasants) are probably all only varieties of *Coccidium tenellum*.

**Natural infection** occurs with food or with other objects taken up by fowl (sand, mud, etc.) and with the drinking water.

Eckardt looks upon coccidia as facultative parasites, which during summer multiply rapidly in remnants of food or other organic material that are taken up accidentally by birds, and which then further develop in their intestines. This view is supported by an observation of Kleinpaul, who saw an affection of ducks which had been kept in a dried marshy swamp.

The spreading of fowl coccidiosis might occur by the aid of eggs laid by sick birds. Eckardt found coccidia on the shells of eggs and in their albumen. The same observations had been made previously by Podvissotzky.

The infection experiments of Railliet & Lucet and Eckardt were positive in 2 or 3 weeks. In Eckardt's experiments symptoms of disease came on within 3 to 6 days and the disease took either an acute or a chronic course.

**Anatomical Changes.** These are found in a portion of the cases in the cecum, and in another in the small intestines, while in other cases the greater portion of the whole intestinal tract is affected. In the acute form of the disease are found changes of an intense acute intestinal catarrh or of enteritis; the intestinal contents are dirty grayish-white, possibly also bloody; the mucosa presents grayish-white nodules or spots up to the size of a millet seed.

Sometimes, particularly in geese, the intestines contain masses of fibrin. In cases of very short duration, the inflammatory changes may be entirely absent, so that the cause of death is only demonstrated by finding under the microscope great numbers of coccidia in the intestinal contents (Kleinpaul).

In subacute or chronic cases the intestinal mucosa of the emaciated animals appears reddened in spots, coated with tenacious mucus and as though dusted with flour. Such dustlike points occasionally occur also in the liver; they are then also due to coccidia (Eckardt).

**Symptoms.** Violent uncontrollable diarrhea, listlessness and increased thirst exist in the **acute form**, while the appetite at first remains normal. Then there occurs profuse salivation, drowsiness, lack of appetite and bluish discoloration of the comb. The droppings become slimy, possibly hemorrhagic. Death occurs after two or four days, but sometimes, particularly in chicks and ducks, suddenly without any premonitory symptoms.

The **subacute form** manifests itself in similar symptoms as the preceding type; its course is, however, slower; the animals remain alive two to four weeks and become emaciated so that they are finally reduced to mere skeletons.

The **chronic form** has been seen in experimental animals and

in natural infection in pheasants; it leads to the development of a marantic condition and diarrhea may alternate with constipation.

According to Eckardt the mortality is 60 to 70 per cent; sometimes all young animals affected die.

**Diagnosis.** The disease can be diagnosticated only by the microscopic discovery of the coccidia in the droppings or in the intestinal contents of dead animals; the described clinical symptoms in connection with the history may excite a proper suspicion. The differential diagnosis has to consider fowl cholera, chicken plague and the various septicemic diseases of birds.

**Treatment.** Morse recommends the administration of 0.006-0.01 gm. of calomel or a few drops of castor oil, with two or three drops of oil of turpentine. Railliet & Lucet prescribe sodium subsulfurosum (0.3-2 gm.) for chickens; Klee for pigeons 5 gm. sulphate of iron and 15 gm. pure glycerine in one quart of boiled water.

**Prophylaxis.** The separation of the sick from the healthy animals must at once be provided for, then thorough disinfection of the coops and daily thorough cleaning. The barnyard where fowls are kept should be kept dry, the droppings should be removed, the incubators should be disinfected and dried out, the floors calcimined. Morse recommends purgation of the hens one or two weeks before they begin laying, and cleaning of the eggs that are to be hatched, with 95 per cent alcohol.

Water fowl should be kept away from half-dried out pools or swamps. Eckardt succeeded in stopping an enzootic of coccidiosis among chicks by given them sterile water and sand sterilized by heat.

**Literature.** Eckardt, B. t. W., 1903, 178.—McFadyean, J. of comp. Path., 1894, 131.—Klee, Geflügelkrkh, 1905, 37; Vet. Jhb., 1906, 352.—Kleinpaul, Pr. Vb., 1904, II, 36.—Martin, Rev. vét., 1909, 413.—Morse, The Vet. Journ., 1908, 389; Pr. Vb., 1901, II, 25.—Railliet, Zool. Méd., 1895, 142.—Zürn, Vortr. f. Tzte., 1878, I, H, 2.

**Perityphlo-hepatitis meleagridum.** (Enter-hepatitis infectiosa meleagridum [Melvin]). Smith in America, Lucet in France and Klee in Germany have observed an epizootic disease of turkeys. It affects animals 2 to 3 months old and has a mortality of 80%. The disease begins with lack of appetite, weakness, and prostration; soon fetid yellowish, thin fluid feces are voided. The animals emaciate rapidly, the comb and the wattles become withered, grayish or dark discolored (hence the English name "black-head"); death occurs at the end of the first or at the beginning of the second week. In less severe cases, the animals improve, diarrhea ceases, comb and wattles regain their turgor and their red color; convalescence is, however, of long duration and recurrences may occur.

The enlarged liver contains numerous yellowish, tough, partly

softened foci. The cecum is uni- or bilaterally enlarged; its wall thickened and covered on the inside by several layers of fibrinous membranes, and the intestinal lumen contains a reddish, ill smelling mass. In the foci in the liver are found highly refractive unicellular bodies, provided with a nucleus, *Amoeba meleagridis*, Smith, which, as shown by the feeding experiments of Melvin, can cause the disease. The disease is due, according to Morse, to organisms identical with fowl coccidia.

Melvin recommends thorough disinfection of the barns, etc., with a watery solution of carbolic acid and sulphuric acid.

**Literature.** Klee, Geflügelkrh., 1905, 38.—Melvin, U. S. Dep. Agric., Circ. 5, 1905.—Morse, The Vet. Journ., 1908, 389.

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## SECTION VI.

### DISEASES OF THE LIVER.

#### 1. Jaundice. Icterus.

**Etiology.** The most frequent cause of jaundice, especially in dogs, is catarrh of the duodenum, with which catarrh of the larger bile ducts (catarrhal jaundice) is often associated. Similarly, jaundice is often a complication of acute gastric catarrh. More rarely, jaundice is caused by impaction of the duodenum and by tumors in the neighborhood of the pylorus (Eberlein). Occlusion of the bile ducts by foreign bodies, parasites (masses of flukes or roundworms), gallstones, neoplasms and fibrous tissue, prevent the outflow of bile. Compression of the bile duct by enlarged portal lymphatic glands, chronic inflammation around the portal fissure, echinococcus cysts, neoplasms and enlargement of other organs may produce the same effect. The passive hyperemia of the liver in both acute and chronic hepatitis causes jaundice by compression of the smallest bile ducts. (Schmidt observed severe jaundice in a horse as a result of volvulus of the left lobe of the liver.)

Thrombosis, compression of the portal vein, owing either to enlargement of the pancreas or of the portal lymphatic glands, and chronic inflammation of the portal vein are very exceptional causes of jaundice.

The immediate cause of the jaundice observed in cases of poisoning by phosphorus, arsenic, salt, lupinose, etc., is to be found in the plugging of the minute primary bile ducts with débris of liver cells.

Not rarely hemoglobinemia (due to piroplasmosis, extensive burning of the skin, and certain infectious diseases, etc.) is associated with jaundice. In such cases excessive quantities of bile pigment are manufactured by the liver cells from the abnormally large amount of blood pigment that is present in solution in the blood plasma (pleiochromia), while there is not a proportional production of the fluid constituents of the bile. The bile produced under these circumstances is viscid and is with difficulty passed out of the liver, consequently a portion is re-absorbed.

The form of jaundice that sometimes occurs during attacks

of acute infectious diseases, severe inflammation of the lungs, and hemoglobinemia arising from unknown causes, was at one time termed hematogenous jaundice. Recent investigations have shown that the bile pigments in these cases also are produced by the liver.

It is not definitely settled whether in individual cases of hepatitis accompanied by jaundice the diseased liver cells do not pass a proportion of the bile direct into the blood capillaries (paracholia, parapedesis).

**Anatomical Changes.** With the exception of the nerve tissue all the organs are stained yellow as a result of the absorption of bile pigments. This is most marked in tissues that contain fat, such as the mesentery, the fat capsule of the kidneys, subepicardial fat and subcutaneous connective tissue. It may, however, also be observed in blood clots, in any fluid exudates that may be present, and in the muscular tissue. (Rüther states that in birds affected with jaundice there may be a greenish pigmentation of the peritoneum.)

In the horse the fat is always yellow. In other animals it is also more or less yellow under certain conditions of diet and even when the animals are fed on certain pastures. In emaciated animals the subcutaneous connective tissue is sometimes saturated at places with yellow serum. The simultaneous yellow discoloration of all the tissues renders the diagnosis of jaundice easy.

**Symptoms.** A characteristic feature of jaundice is the yellow coloration of the mucous membranes and of the unpigmented parts of the skin. This is recognizable in the earliest stages in the scleral and palpebral portions of the conjunctivæ. It is less distinct in the mucous membranes of the mouth and nose, unless one causes a temporary anemia of the membrane by exerting pressure upon it. The color varies with the severity and duration of the disease from a lemon- to an orange-, or even a greenish yellow tint. Unpigmented parts of the skin appear, as a rule, rather more intensely stained.

The condition may be recognized still earlier by an examination of the urine which is either orange-, brownish-, or greenish-yellow in color, and if agitated gives a yellow froth. White filter paper, linen or silk are stained by the urine. Chemical tests show the presence of bile pigments and often also of bile acids, while microscopic examination of the sediment reveals the presence of masses of pigment which give the bilirubin reaction.

Gmelin's test is one of the best for the chemical demonstration of the presence of bile pigments. In this test the suspected urine is added to some nitric acid containing a few drops of nitrous acid so that it forms a layer above it. A positive result is indicated by a play of colors where the liquids meet, green being the most characteristic. Rosenbach's modification of this test appears to be a practical one. A piece of filter paper moistened with the urine, or better still the paper through which the urine has been filtered, is placed upon a white porcelain slab and is touched with a glass rod that has been dipped in fuming nitric acid. If bile pigments are present a ring of colors appears around the spot where the acid has been placed. These colors from within outwards are yellow, violet, brown, green. In some cases only the green color is obtained.

flammatory lesions about the portal fissure cause a more lasting and serious condition.

**Diagnosis.** In view of the fact that jaundice is merely a symptom of various organic diseases, the cause of the engorgement with, or absorption of bile must be thoroughly investigated.

**Treatment.** As a rule good results follow treatment similar to that adopted in gastric catarrh (see page 291).

In addition to careful regulation of the diet, from which all fat is rigorously excluded, the administration of a course of neutral salts is advisable. For dogs a 10 per cent lukewarm solution of Carlsbad salts may be employed. Frequent flushing of the rectum with large quantities of water has a beneficial effect. In order to dilute the bile and to stimulate the contractions of the gall bladder, bile ducts and the intestine, Cozessa advises the subcutaneous injection of pilocarpine in doses (for dogs) of 0.005 to 0.01 gm., followed by two or three doses of 5 gm. of potassium tartrate. Mild purgatives, such as calomel and aloes, are indicated where there is constipation, and in cases in which there is oliguria diuretics such as lemon juice and potassium acetate or nitrate. According to Siedamgrotzky, faradization of the liver is beneficial (?). Horneck has had good results from the intravenous infusion of physiological salt solution.

**Literature.** Balás, Á. L., 1907, 17.—Bierthen, D. t. W., 1906, 481 (Lit. on the chem. demonstration of bilirubin).—Chierici, N. Erc., 1908, XIII, 420.—Kohnhäuser, Monatsschr. österreich. Tzte., 1881, 84.—Latschenberger, O. Z. f. Vk., 1887, 47.—Siedamgrotzky, S. B., 1883, 17.

**Icterus Neonatorum.** New-born animals, especially calves and foals, sometimes show symptoms of jaundice a few days after birth and exceptionally they are diseased at the time of birth. Sometimes the jaundice disappears together with the derangement of digestion in the course of a few days. In the majority of cases the animals quickly become exhausted and very soon die. Hartmann, who frequently observed the disease in the Bábolna Stud, advised the administration of magnesium carbonate and rhubarb.

The cause of the condition is unknown. Hartmann's view, that the jaundice results from inflammation of the umbilical veins, may be correct in rare instances. Probably there are several factors in the production of the disease. In the first place an extensive destruction of red blood corpuscles may lead to a condition of pleiochromia, (see page 506). Secondly, some of the bile-pigments which are not decomposed in the intestine may be absorbed into the branches of the portal vein and pass directly by way of the ductus Arantii into the posterior vena cava, and thence into the general circulation. The venous congestion due to debility and imperfect respiration must also be taken into consideration (Birch-Hirschfeld, Johnne).

French authors (Levrier, Bernadin, and others), have described a disease of mules, and Dieckerhoff, a disease of foals, which is an acute infectious malady complicated with jaundice and not uncommonly with hematuria. It is not impossible that this is a severe pyemia and



septicemia of umbilical origin. The name given by these authors to this condition is *icterus gravis*.

**Literature.** Dieckerhoff, *Spez. Path.*, 1904, I, 1031.—Hartmann, *Ö. Vj.*, 1880, LIII, 2.—Schöttler, *A. f. Tk.*, 1898, XXIV, 297.

## 2. Gallstone Disease. Cholelithiasis.

**Occurrence.** Apart from precipitations and incrustations in the bile ducts, true gallstones are of rare occurrence. They are, however, found most frequently in the horse and ox, more rarely in the dog, and exceptionally in the cat, pig and sheep. Kitt found a calculus in the gall bladder of a fowl.

**Etiology.** The most frequent cause of precipitation of the bile salts is catarrh, either of the bile ducts or gall bladder. The large amount of mucus secreted acts upon the bile and especially on the salts of the bile acids and decomposes them, while a large quantity of cholesterin is added to the bile through the desquamation of the epithelial cells. The cholesterin and the bile pigments are held in solution by the bile acids. The decomposition of the acids leads to the precipitation of the cholesterin and pigments. At the same time carbonate of lime is precipitated or else it is produced by the disintegration of the epithelial cells shed from the diseased mucous membrane (Naunyn). Partial or complete stasis of the flow of bile tends to the formation of biliary calculi, and this is also likely to cause catarrhal inflammation.

In the livers of ruminants there are frequently soft masses of precipitated bile salts covering the inner surface of the bile ducts to a variable thickness and also mixed with the bile. In old animals these masses form hollow cylinders lining, and firmly adherent to, the inner surface of the walls of the bile ducts. This is frequently the case in distomatosis. On the other hand, in horses it is possible that the cause is a bacterial infection derived from the duodenum which leads to catarrh of the bile ducts.

In the majority of cases the starting point of a true gallstone is a mass of desquamated epithelium, mucus and bacteria, around which the precipitated bile salts and cholesterin are deposited in layers. A similar part is played by dead animal parasites, such as flukes, or their eggs. Occasionally, the starting point may be a foreign body which has passed into the bile duct from the duodenum. Since stagnation of the bile favors the deposition of the bile salts and thus aids bacterial infection, gallstones are more frequently found in animals that are always stabled or move about but little.

**Anatomical Changes.** In the horse and ox the bile ducts sometimes contain hard cones or cylinders which may be as thick as a finger. In the fresh state they are light or dark

brownish-yellow in color and smooth, and when dried, rough and laminated (Kitt). In other cases they are white and composed of chalk-like masses containing minute particles of food. Gurlt found such a stone weighing 5 pounds.

True gallstones are usually found in larger numbers in the principal bile ducts, or more frequently in the gall bladder. They are either rounded with mulberry-like rough surfaces or polyhedra with smooth facets, yellowish-brown, greenish, gray or reddish-brown in color. The majority are not very hard and have a specific gravity slightly higher than that of water (1.23). Their cut surfaces shows laminæ of different colors enclosing a more or less soft center. The inner layers are composed principally of cholesterin, and the outer of bile pigments and calcium salts. Gallstones obtained from cattle are generally lighter and contain a greater proportion of organic matter. In the process of drying they may shrink to half their original size.

Small stones varying in size from a linseed to a pea are sometimes found in very large numbers. Birnbaum found 400 in the bile ducts of a horse, Rigot 90, Avérous more than a thousand, Lewin 500 to 600. Single stones, on the other hand, sometimes attain the size of an apple (Verheyen). In Avérous' case there was, in addition to the numerous small stones, a large one measuring  $11\frac{1}{2}$  cm. long and weighing 258 gr. in the terminal portion of the principal duct. Messner found a stone weighing 542 gr. in an ox.

The chemical composition of the stones varies from case to case, and especially with regard to the quantity of cholesterin. In gallstones obtained from horses Fürstenberg found lime, mucus, fat and bile pigments. In cattle the stones contain a comparatively large proportion of carbonate and phosphate of lime and magnesium. Bourgoin found a stone weighing 330 gr. in the bile duct of a horse. In this case the stone was composed entirely of cholesterin.

Impaction of gallstones in the common bile duct leads to the production of an inflammatory, hyperplastic cirrhosis of the liver. Occasionally abscess formation results. In other cases there is dilatation of the bile duct associated with atrophy of the surrounding liver tissue. In cattle the common bile duct sometimes attains a diameter equal to that of the large intestine. In such cases peritonitis due to the rupture of the dilated ducts may be the immediate cause of death (Cagny, Chaisaing).

According to Lewin gallstones are sometimes found in the intestine, these having been passed out of the bile ducts.

**Symptoms.** The gall bladder may contain a large number of gallstones without there being any disturbance of the animal's health. Eberhard found the gall bladder quite full of stones in a pig that was apparently in perfect health. Since gallstones obstruct the outflow of bile the chronic symptoms to which they give rise are not characteristic. The animals usually have a capricious appetite. Offensive diarrhea and constipation alternate. In ruminants there is tympanites, dullness, gradual wasting and anemia, without any symptoms enabling one to determine the cause of the condition.

The sudden impaction of a large gallstone in the common duct or in the neck of the gall bladder produces cramp-like contractions of the wall of the duct. In this way horses and occasionally cattle are attacked with colic-like pains (gallstone colic). These pains may persist with remissions for some days and then disappear. The colic is sometimes associated with loss of consciousness (Birnbäum, Lewin).

On the second or third day of the attack, and in some cases earlier, there is biliary pigmentation of the mucous membranes which appears suddenly with the onset of pain, and disappears as quickly when the pain ceases. In cases in which the neck of the gall bladder is obstructed there may be no evidence of jaundice. During the attacks of colic there is a rise of temperature. The pulse may be either rapid and small or slow and full, and not rarely irregular. In addition to the constipation and diarrhea vomiting is seen in the dog. Wyssmann observed symptoms of hemorrhagic nephritis in a horse.

The simultaneous occurrence of colic, jaundice, great depression, and weak pulse affords the best indication as to the nature of the condition, especially when the attacks of colic are repeated at intervals. The sensitiveness to pressure and the enlargement of the liver which can often be demonstrated in these cases are of value for diagnosis. If the gallstone does not move further, the jaundice gradually increases until the patient finally dies showing symptoms of cholemia (see page 567.) Rupture of the gall bladder is followed by sudden collapse, the animal dying in the course of one to two days from acute peritonitis (Mollereau & Cagny). In a cow that had died suddenly, Kohlhepp found intestinal contents in the greatly dilated gall bladder.

**Treatment.** During the attacks of colic, narcotics, such as morphia, injected subcutaneously, chloral hydrate per os or per anum, and inhalation of chloroform are indicated. Mild purgatives, such as olive oil, are likely to assist in moving the stone by causing an immediate increase in the flow of bile. Parascandolo removed a gallstone from the bile duct of a dog by laparotomy, recovery being established in four weeks.

For the prevention of the formation of fresh stones, moderate diet and plenty of exercise associated with an extended course of neutral salts are indicated.

**Literature.** Eberhard, B. t. W., 1905, 116.—Fröhner, Monh., 1894, V, 61.—Kohlhepp, B. Mt., 1905, 100.—Lewin, Z. f. Vk., 1905, 62.—Parascandolo, A. f. Tk., 1902, XXVIII, 484.—Trolldenier, Monh., 1904, XV, 193 (Lit.).—Wyssmann, Schw. A., 1906, XLVIII, 89 (Lit.).

**Foreign Bodies in the Liver and Bile Ducts.** Occasionally pointed foreign bodies perforate the reticulum or abomasum and penetrate into the liver. This as a rule leads to the formation of an abscess in the liver and the animal presents the symptoms of traumatic gastritis and purulent hepatitis. Saint-Cyr found the straw of a leguminous



plant in the liver of a horse. Mégnin records a case in which he found some awns of barley which had obviously come from either the stomach or duodenum lying lengthwise in the gastro-hepatic ligament. In the first case, the straw perforated the portal vein and caused thrombosis, and in the second, death was due to hemorrhage. On three occasions Cadéac and Blanc found a needle in the liver of dogs. One of these showed symptoms resembling those of rabies.

In the bile-ducts of oxen, pigs and horses large quantities of sand are sometimes found. This is due to the ingestion of dirty food. In a case recorded by Augenheister the greatly dilated bile-ducts contained 10 lbs. of sand.—(Guillebeau, Schw. A., 1900, XLII. 248.—Müller, S. B., 1903 260.)

### 3. Fatty Liver. Hepar adiposum.

#### *(Fatty Degeneration and Infiltration of the Liver.)*

**Etiology.** The following factors may lead to an increase in the fat content of the liver: Very rich diet in the process of fattening, which is best seen in birds, insufficient exercise, diminished oxidation in the body brought about by anemia. On the other hand, the liver often becomes fatty when there is sudden wasting of an animal as in diabetes mellitus and in certain diseases which run a rapid course. In these cases large quantities of fat are reabsorbed from the fat-containing tissues by the liver, and then gradually consumed.

In many cases fatty liver is caused by noxious chemical materials which are either absorbed from the alimentary canal or are circulating in the blood. The principal of these are the bacterial toxins, fatty liver being a frequent lesion in acute infectious diseases. Acute yellow atrophy of the liver is apparently due to bacterial toxins. Certain poisonous plants produce the same effect. Fatty liver may be associated with gastroenteritis, due to the ingestion of mouldy fodder. The effect of lupines in this connection is remarkable. Of the mineral poisons arsenic, antimony, lead and phosphorus are the principal causes and especially the latter. The chief organic compounds that cause fatty liver are carbolic acid and alcohol.

**Pathogenesis.** Recent investigations have shown that in the process of fattening, obesity, sudden emaciation and in poisoning by various substances the fat is brought to the liver either from the food material or from the adipose connective tissues of the body. The noxious chemical substances cause a sudden and extensive disintegration of the tissues with the result that a large amount of the reserve body fat is in a condition for absorption and this is stored up for a time in the liver. These substances also have an effect upon the liver cells rendering them inactive and thus unable to deal with the fat brought to the liver or to oxidize the partly synthesized fat.

The disturbance of the functions of the liver, owing to the

presence of a large amount of stored up fat, is greatest after there has been a very large quantity stored. Where the fatty condition of the liver is due to chemical substances there is a derangement of function and of nutrition of the liver from the very outset, this being due to the action of the chemical substance and not to the fat. In view of the fact that these substances cause considerable disintegration of the protein substances of the cells it is possible that a portion of the fat is derived from the disintegrated proteins. Many authors deny this possibility (F. Müller, Pflüger).

It was at one time customary to describe two forms of fatty liver; fatty infiltration, in which the fat was brought to the liver from without, and fatty degeneration in which there was an actual change in the protoplasm of the cell resulting in the production of fat. This distinction is now no longer possible. It would be better to distinguish between a process whereby the liver becomes fatty that is associated with destruction of the liver cells and one in which the liver becomes fatty that is not associated with any cell destruction.

Rosenfeld fed dogs experimentally for long periods with mutton suet and after a period of starvation poisoned them with phosphorus. Chemical analysis proved that the fat contained in the liver was mutton fat. Fowls that had been starved until they had lost practically all their fat showed no fatty changes in the liver when poisoned with phosphorus.

**Anatomical Changes.** The liver appears pale-yellow and in severe cases may be as yellow as butter or ochre. In the early stages the peripheral parts of the lobes are yellow. In advanced cases the entire liver is intensely yellow in color. The absence of a greenish tint excludes the possibility that the discoloration is due to bile. The liver is enlarged (Neyraud saw a horse's liver that weighed 14 kilos; and Kitt, a pig's liver weighing 14.8 kilos), the edges are rounded, the consistency is decreased, it feels greasy to the touch and pits made by pressure with the finger persist. On cutting into it the blade of the knife is found to be covered with a layer of fat. In cases in which the protoplasm of the liver cells is involved the liver may be actually smaller than normal on account of the absorption of the destroyed liver cells. Evidences of this destruction can be seen under the microscope.

**Symptoms.** A fatty condition of the liver may be suspected when the predisposing factors are in operation and the liver is enlarged without there being any other symptoms of disease of that organ. When the liver is enlarged there is an increased area of hepatic dullness, and the edges of the organ can be palpated. If the animal be very fat it is generally impossible to ascertain whether the liver is enlarged but the general condition of the animal makes it extremely likely that the liver is fatty. In the horse, digestive disturbances and particularly constipation are generally observed.

Occasionally fatty livers rupture and the animals die suddenly as a result of internal hemorrhage. There is sometimes slight abdominal pain (Neyraud). Neale records a number of cases in Shropshire sheep which ended fatally after a short illness and in which the only lesion found at the postmortem was extreme fatty infiltration of the liver. Rupture of the liver is especially frequent in well-nourished fowls and geese (Johne).

**Treatment.** The treatment must be adapted to the nature of the case.

**Literature.** John, S. B., 1879, 49.—Neyraud, J. Vét., 1892, 400.—Rievel D. t. W., 1906, 49 (Lit.).

#### 4. Amyloid Disease of the Liver. Degeneratio amyloidea hepatis.

(*Hepar amyloideum, Amyloidosis hepatis.*)

**Etiology.** Amyloid degeneration of the liver and other organs occurs usually in protracted, exhausting diseases. In the horse, in which animal it is most frequently seen, it occurs in cases of chronic inflammation of the serous membranes, particularly of the pleura.

As regards the frequency of the condition in the horse, Bohl found amyloid liver in 4 per cent of all the horses examined postmortem. Rabe found amyloid degeneration of the liver in practically 50 per cent of horses that had suffered from chronic diseases of the serous membranes. The degeneration also occurs in cases of bronchial catarrh, chronic interstitial pneumonia, glanders and, exceptionally, occlusion of the bile ducts. The observation of E. Noyer and Grüner regarding the special part played by certain infectious diseases in the production of amyloid liver are very interesting.

E. Noyer observed amyloid degeneration of the liver followed by rupture of that organ in about 20% of horses used for the production of diphtheria serum. Grüner found amyloid liver in every one of 46 horses that had died from contagious pleuro-pneumonia. On the other hand the very rare occurrence of amyloid liver in the domesticated animals has been recorded by Joest, Pflug, Förster, Paulicky and Hissbach.

Rabe observed the condition in a bitch in connection with carcinoma of the mamma; Bruckmüller, in cattle, as the result of tuberculosis and chronic nephritis, and Ries in lymphangioitis. Chronic suppuration, especially when it involves bone, and chronic abscesses may also cause amyloid degeneration.

Attempts have been made to clear up the etiology of amyloid degeneration by experimental investigation. The first experiments were made by Krawkow, who gave dogs, rabbits, fowls and pigeons repeated subcutaneous inoculations of cultures of the *Staphylococcus pyogenes aureus*. Pronounced amyloid degeneration was produced and especially in the liver. The degeneration was also produced when the chemical substances produced in the cultures were used for the inoculations, and in one case the systematic introduction into the body of the toxins of the bacillus pyocyaneus was followed by amyloid disease. The experiments were repeated by



Maximow with similar results and Davidsohn's results with rabbits, guinea pigs, mice and fowls were also positive. Lubarsch was able to produce amyloid degeneration in 3 or 4 weeks in rabbits and dogs by systematic subcutaneous injections of oil of turpentine (suppuration!).

The nature of the change in amyloid degeneration has not been definitely settled up to the present nor has it been determined how the amyloid material, which possesses some of the characters of proteins, is produced. It is not known whether the material is elaborated in the liver or whether it is brought to the liver by the blood. The former view appears to be the more probable, the theory being that the cells are in a cachectic condition and are therefore unable to deal with the proteid materials, which are consequently left in the tissue spaces and become converted into amyloid material (Wichmann). On the other hand the observations of Noyer and Grüner indicate that amyloid degeneration is the result of injurious effects exercised upon the tissue cells by certain poisons.

**Anatomical Changes.** The liver is the organ that is most commonly affected. In cases where the degeneration is extensive the liver may be enlarged three to four times. The edges appear thick and rounded, and the capsule is often considerably thickened. The color varies from light brown to yellow or gray. The lobulation is distinct because the peripheral part of each lobule is pale grayish-red and lardaceous, the central portion being pale brownish-red, grayish-brown, or occasionally yellow. The consistency is more or less doughlike, and in the later stages crumbling like half-dried mortar. In birds amyloid liver is granular and brittle. In the early stages no abnormalities save slight enlargement, distinct lobulation and pale color are observed.

Amyloid material may be demonstrated in affected organs by its color reactions. The application of Lugol's solution for a few minutes to the cut surface stains the degenerated parts mahogany-brown. The same test may be applied to microscope sections, the color being changed to a dirty violet or bluish-red by the addition of 2% sulphuric acid. Safranin gives an orange-yellow color.

In the horse the amyloid material is deposited in the outer zone of the lobules and gradually extends inwards towards the center. In this way the connection between adjacent lobules is soon destroyed and thus the softening of the organ is produced. The amyloid material is deposited in the walls of the capillaries and in the intima of larger vessels.

**Symptoms.** In cases where there is or has been some primary diseased condition which experience has shown is likely to be followed by amyloid degeneration, a diagnosis of this condition may be based upon the increased area of dullness in the region of the liver and upon palpation of the liver. The symptoms are obscured by those of the primary disease, but as a rule, such symptoms as anorexia, irregular defecation, dullness, wasting and albuminuria, may be due to amyloid disease itself.

The disease is rarely associated with ascites or jaundice. Rupture of the softened liver and resulting hemorrhage are often observed whether there be any traumatic influence in operation or not. In addition to anorexia and dullness, Rexante observed an intermittent bright yellow coloration of the feces which were passed in small quantities only. The horse also had slight attacks of colic and yellowness of the mucous membranes.

**Treatment.** As the condition is always secondary, attention must be paid chiefly to the treatment of the primary disease.

**Literature.** Bohl, Arch. f. Vet.-Wiss., 1905, 236.—Davidsohn, V. A., 1897, CL, 16 (Lit. on experim. amyloid degen.).—Grüner, Archiv. f. Vet.-Wiss., 1906, 740.—Hissbach, Leipz. Ber., 1906, 13.—Joest, Ergebn. d. Path., 1907, XII, 444 (Lit.).—Luharsch, ibid., 1897, IV, 449 (Lit. on experim. amyloid degen.).—Noyer, Über Leberblutung inf. Amyloiddeg. b. Pferd. Diss. Bern, 1904 (Lit.).—Wiktorow, Zur Frage üb. d. amyl. Entart. der Leber u. d. Milz b. Pferd. (Monographie; russ.), 1905.

## 5. Rupture of the Liver. Ruptura hepatis.

(*Hæmorrhagia s. Apoplexia hepatis.*)

**Etiology.** The comparative richness in blood and the softness of the liver tissue explain the rupture of even healthy livers when subjected to some sudden mechanical influence which causes a rise of internal pressure. Among such influences may be included jumping, falling, kicking, severe vomiting, etc. (Johne). Rupture of the liver may be caused by fractured ribs. In cattle foreign bodies passing through the stomach wall may also cause this accident. Parasites, such as the *Pentastoma denticulatum* in the sheep, fluke and *Cystercercus tenuicollis* in ruminants and pigs, have also been responsible.

As a predisposing cause of rupture of the liver, there is usually a degeneration of some kind of the liver tissue. In this connection, at least as regards the horse, amyloid disease is the most important. Parenchymatous degeneration and more frequently fatty degeneration, abnormal conditions of the blood vessels in acute infectious diseases such as anthrax, purpura, hemorrhagic septicemia, hepatic lesions of a glanderous or tuberculous nature, the latter especially in birds, abscesses (Schüler) and neoplasms (angioma, sarcoma, carcinoma) sometimes determine rupture of the liver. Elevations of blood pressure, such as occur in the early stages of inflammatory processes and in the veins of the liver as a result of diseases of the heart and lungs, are predisposing causes of hemorrhage of the liver. Hemorrhage may also follow embolism of the hepatic artery or portal vein. In all these instances the hemorrhage occurs either spontaneously or in response to some trifling external influence.

According to Wörz too liberal a diet may cause parenchymatous hemorrhages in the liver, the over-filled stomach and intestines causing pressure on the liver and portal vein and thus producing congestion of the vessels of the liver. As mentioned by Noyer, rupture is very likely to occur in such cases when the liver is amyloid.

**Anatomical Changes.** In cases where there is rupture of a large number of small vessels the liver is beset with small dark punctiform hemorrhages. Such hemorrhages and even large ones may later become enclosed in fibrous capsules. These



sometimes become calcified (Bruckmüller), or, after absorption of the granular débris, converted into cysts with red-tinged serous contents. On the other hand, old hemorrhages may be indicated by fibrous cicatrices only.

In large hemorrhages, such as commonly occur in amyloid livers, there is extensive breaking down of the liver tissue, forming large cavities with soft irregular walls and containing coagulated blood (hematoma). In such cases the weight of the liver is increased. In a case reported by Trasbot the liver of a horse weighed 33 kilogrammes. When the superficial layers of the liver tissue are broken through, the blood collects under the serous covering and elevates it. In these cases, there is a cavity filled with blood, immediately under the serous membrane. By the bursting of such hematomata large quantities of blood may be set free into the peritoneum. After extensive hemorrhage either into the liver tissue or into the peritoneum, there is evidence of severe anemia of all the other organs.

**Symptoms.** Small hemorrhages into the liver tissue cause no symptoms. If the hemorrhage is severe, symptoms of internal bleeding are produced. The animal suddenly becomes dull, it has an anxious expression, the pulse is accelerated and weak, the mucous membranes are pale and dry and the peripheral parts of the body are cold. Soon after, there is local or general sweating, muscular tremors and staggering follow, and finally the animal dies in from one to ten hours with convulsions.

In cases where the hemorrhage is not so profuse, the symptoms are less severe. In many cases the only abnormalities observed are weakness, acceleration of the pulse, inappetence, jaundice, slight constipation, stiffness of gait, susceptibility to pressure over the liver or extension of the area of hepatic dullness. The latter symptom has been observed by Weber in a number of cases in the horse. In one particular case in a horse, a hematoma in the neighborhood of the portal vein caused marked swelling of the spleen. In some such cases there is improvement, at least for a time, in others, there is a sudden wasting and death in five to six days.

In a case recorded by Dieckerhoff a horse lost so much blood that it was incapable of work for 3 or 4 months, but afterwards worked in a wagon for a year.

In the cases recorded by Wörz in which there were repeated hemorrhages, the horses at first showed temporary digestive disturbances and slight abdominal pain. Later there was anemia, dullness, wasting, edema of the legs, under surface of the body and sheath. These symptoms were repeated with gradually increasing severity, the dullness and depression became very pronounced, the feces were dry and mixed with large quantities of imperfectly digested food and the urine was dark brown in color. Finally the animals died in a state of complete exhaustion, if a rupture of the liver did not supervene and kill them.

**Treatment.** When there is a suspicion of hemorrhage of the liver the most important factor in the treatment is absolute rest. Ergotin, extract of hydrastis or adrenalin may be



used. The dose of the latter for a horse is 1 to 5 cc. of a 1:1000 solution per 100 kilograms body weight; and for dogs, 0.5 cc. The drugs are administered by subcutaneous injection. The constipation should be relieved by enemata, and where there is great weakness of the heart, camphor and caffeine are indicated.

**Literature.** Bergeon, *Rev. vét.*, 1905, 243.—Johne, S. B., 1879, 49.—Noyer, *Über Leberblutung infolge Amyloiddegen. b. Pferd.* Diss. Bern, 1904 (Lit.); *Pr. Mil. Vb.*, 1899-1906.—Zündel, *Z. f. Vet.-Wiss.*, 1874, 307.

## 6. Acute Parenchymatous Hepatitis. *Hepatitis parenchymatosa acuta.*

Under the term acute parenchymatous hepatitis, are included inflammatory diseases of the liver substance in which, in addition to cellular infiltration and hyperemia, there is pronounced cloudy swelling and fatty infiltration of the liver cells.

**Etiology.** Acute hepatitis is rarely observed as a primary condition. It is generally caused by poisonous substances or by bacteria which reach the liver by way of the portal vein from the intestine. Possibly the organisms sometimes reach the liver by way of the bile duct. Apart from those caused by defective food or poisonous plants, cases of hepatitis sometimes occur in association with catarrh of the stomach and intestine, but in which the liver disease is apparently primary. Two such cases are recorded by Fröhner in which there were no lesions of any importance in any of the other organs.

The two cases of fatal acute hepatitis described by Kas and Lipa in the horse were probably cases of amyloid disease. Robertson and others have stated that certain plants belonging to the genus *Senecio* have in some cases produced acute hepatitis associated with catarrh of the stomach and intestine, but that the lesion so caused is usually in the nature of a chronic interstitial hepatitis.

As a rule, acute hepatitis is purely secondary to some acute disease. It may be seen in gastro-enteritis, or it may be caused by certain poisons, lupinose, phosphorus, arsenic, etc. The symptoms are generally obscured by those of the primary disease and consequently the clinical aspect of the condition is of little importance. Not rarely acute hepatitis is set up by the migration of certain animal parasites (distomes, cysterici, pentastomes, sclerostomes). This type of hepatitis will be considered under a separate heading.

**Anatomical Changes.** The liver is enlarged and its edges are rounded. The cut surface is dull and the glandular arrangement is obscure, but small reddish patches or dark red hemorrhages may be present. Its consistency is soft and crumbling.

**Symptoms.** Susceptibility to pressure over the region of the liver, evidence of pain during defecation, and finally jaun-

dice indicate acute hepatitis, provided the history of the case is in agreement with that. Diagnosis can be made earliest in the dog. Fröhner observed two cases in which, besides great weakness, there was severe jaundice and susceptibility to pressure over the liver. In one case there was fever.

In the acute hepatitis caused by the plants of the genus *Senecio*, there is persistent diarrhea, marked tenesmus and frequently prolapse of the rectum. There are also symptoms of excitement, and death occurs in 2 to 3 days.

**Treatment.** The line of treatment indicated is the administration of neutral salts, various mild purgatives or disinfectants. The quantity of fat in the diet should be restricted, and the diet should be changed.

**Literature.** Fröhner, *Monh.*, 1894, V, 306.—Kas, T. Z., 1906, 249.—Lipa, *ibid.*, 1907, 289.—Robertson, *J. of Comp. Path.*, 1906, 97.

**Acute Diffuse Parenchymatous Hepatitis of the Sheep.** Azary records the annual occurrence during the months of January and February of a peculiar disease among the pregnant ewes on a farm in Hungary. During a period of five years the losses amounted to 473 ewes (29% of the flock). The disease broke out a few days after the animals were brought in, and attacked principally the ewes that were in the most advanced stage of pregnancy, and of these only the best nourished.

The symptoms presented were as follows: The animals became markedly dull and lay on the ground with half-closed eyes. On the second or third day, after a partial recovery, rumination was suspended and the animals again became semi-comatose. The temperature was normal at first, but then rose to 40°-41.5° C. before death. At the outset the pulse was accelerated and later became weak. Respiration was hurried throughout, and in the late stages was of the Cheyne-Stokes type. The animals lay on their sides seeking to relieve the pressure over the region of the liver. The urine was reduced in quantity and contained albumen, epithelial cells from the straight tubules of the kidneys and from the bladder, and leucin bodies. On the 5th or 6th day, there was complete apathy, grinding of the teeth and muscular tremors. The wool came out in large tufts and the animals died about the end of the first week in a state of complete exhaustion.

At the postmortem the liver was found to be enlarged, anemic, more or less soft in consistency, yellowish or exceptionally brownish-yellow in color, as in cases of poisoning by phosphorus. The lobules were grayish-yellow and translucent, with a grayish-white peripheral zone. The margins of the lobules were indistinct. Besides numerous droplets of fat the liver cells contained rounded yellow translucent bodies that were not soluble in alcohol or ether (leucine?). There was no evidence either of parenchymatous or fatty degeneration in the kidneys but the above-mentioned bodies were present in the lumen of the tubules. Other lesions were: hemorrhages of the serous membranes, and parenchymatous degeneration of the heart-muscle.

Azary described the disease as an acute diffuse parenchymatous

hepatitis which showed considerable resemblance to the yellow atrophy of the liver seen in fashionable women during the last months of pregnancy. The cause of the condition could not be determined.

A similar disease was observed a year later by Czakó & Hartmann. In this case, there was no fever and the disease took a benign course. Flóris has recently recorded a disease occurring in Hungary, among ewes that had been kept indoors for weeks. He connected the disease with the number of fetuses in the uterus of the ewes, as in every case that came under his observation there were three.

A similar disease was described by Murillo & Izcara as an infectious inflammation of the bile-ducts and gall-bladder. This was referred to a bacterial invasion.

Haubold records a disease occurring among sheep during the autumn months, the symptoms of which were: loss of appetite, great weakness, increased thirst, jaundice, soft, pultaceous, and, at the last, blood-stained feces. The cause was probably the exclusive use of wet grass and clover, for no further symptoms were observed after the diet was altered.

According to Ömler, the following set of symptoms were seen either on the same or the following day, in a flock of sheep feeding off fields of good rye stubble; great weakness and somnolence, inappetence, watery diarrhea that was rather offensive and sometimes mixed with blood, staggering gait, pain on pressure over the abdomen, hurried respiration, fever, purulent discharge from the nose that was sometimes blood-stained, rather bright red coloration of the mucous membrane of the mouth. One-third to two-thirds of the animals died within 1 to 2 days. In addition to inflammatory changes in the stomachs and intestines, the liver was found to be grayish-yellow in color and easily torn.

**Literature.** Azary, Vet., 1883, 69.—Flóris, A. L., 1907, 497.—Hartmann, Ö. Vj., 1884, LXI, 181.—Haubold, S. B., 1888, 75.—Ömler, A. f. Tk., 1883, IX, 210.

**Enzootic Hepatitis of Young Pigs.** Semmer has recorded an enzootic among two-months-old pigs that caused great losses in Russia. In these cases the liver was enlarged and nodular. The cut surface presented a variegated mosaic-like appearance, dark red patches alternating with bright red or grayish-yellow areas. The peritoneum and sometimes the pleura contained a serous exudate, the intestines were inflamed, the urine contained albumen. Between the liver cells, which were infiltrated with fat, there were small-celled infiltrations and collections of extravasated red blood corpuscles. The only symptoms observed were debility and inappetence just before death. In the liver, spleen and blood, Nonewitsch found large cocci, cultures of which when inoculated into young pigs caused death in 7 to 8 weeks. At the post-mortem the above-described lesions were found. From the long period elapsing between inoculation and death and from the fact that only pigs of about 2 to 4 months old were attacked, the author concludes that the infection occurs immediately after birth, possibly by way of the navel.

According to Kleinpaul, Brädel and Willerding the disease occurs in Eastern Prussia and it has recently broken out in a very virulent form. During the year 1906, the losses were greater than those due to swine plague. According to the histological investigations of Brädel, who describes the disease as hepatitis haemorrhagica mortis-



feans, the condition is not due to cysticerci or distomes as supposed by Seiler and Wolffhügel. Brädel also points out that the hepatitis may be connected in some way with swine fever.

**Literature.** Brädel, Ein Beitr. z. Kenntn. d. Leberkrankh. d. Schweines. Diss. Giessen, 1908 (Lit.).—Kleinpaul, B. t. W., 1907, 101.—Nonewitsch, Cbl. f. Bakt., 1888, III, 233; Ö. M., 1890, 440 (Rev.).—Seiler, D. t. W., 1907, 436.—Semmer, Ö. Vj., 1874, XLI, 136.—Willerding, Pr. Vb., 1908, II, 58.—Wolffhügel, Z. f. Infkr., 1907, II, 546.

## 7. Acute Yellow Atrophy of the Liver. *Atrophia hepatis flava.*

**Etiology.** Poisoning by certain substances, such as phosphorus and lupinose (Schneidemühl), is followed by a sudden and extensive fatty degeneration associated with destruction of the liver cells. This destruction of tissue leads to a reduction in the size of the organ. The same cause is probably in operation in those cases which have come under observation following the ingestion of sour potato peelings (Haubner), pea or vetch straw (Reinemann, Jansen), and green vetches (Stöhr). Occasionally, such cases occur without a discoverable cause. In some cases the coincident existence of septicemia or gastro-enteritis suggests the possibility of a toxin being the cause. In other instances poisons contained in the food may be responsible. Haubner and Franze saw cases following the use of hay from flooded land.

The disease has been observed principally in the horse and sheep. Callot records an epizootic among cattle in Uruguay.

**Anatomical Changes.** The liver is reduced in size, corrugated, very soft and ochre-yellow in color. The cut surface from which a large quantity of fatty debris can be scraped, shows at places somewhat firmer red areas in which the liver cells are for the most part destroyed, and the vessels are dilated. Besides a cellular infiltration of the liver tissue, the epithelium of the bile ducts appears in some cases to be degenerated, while the smallest bile ducts have proliferated. There is fatty degeneration of the kidneys. At the postmortem, there may also be found enlargement of the lymphatic glands; and of the spleen, catarrh of the stomach and intestines and hemorrhages in other organs.

**Symptoms.** The onset of the disease is marked by sudden depression and weakness, fever, jaundice of the mucous membranes, inappetence and, in some cases, attacks of colic with tenderness of the abdomen (Zündel). In the later stages there is diarrhea. The jaundice and dullness soon become very marked, but at intervals there are symptoms of excitement.

As a result of the coincident disease of the kidneys, the urine is diminished in amount, reddish-brown in color and con-

tains bile pigments and albumin. In the human subject leucin tyrosin are sometimes present in the urine. On the addition of acetic acid these are precipitated in the form of rounded masses and needlelike crystals.

Death usually takes place in about six or eight days. The reduction in the amount of urine and the presence of albumin in it are unfavorable signs.

French authors (Cruzel, Beauvais, Delage), have described an acute hepatitis in cattle in which the liver is enlarged and clay-colored. The symptoms are general malaise, inappetence, salivation, grinding of the teeth, staggering gait, jaundice, pain in the right side of the abdomen and constipation followed by diarrhea.

**Treatment.** Disinfectants, mild purgatives and diuretics may be tried, but so far all treatment has proved useless.

**Literature.** Adam, W. f. Tk., 1857, 3.—Beauvais, Rev. vét., 1894, 497.—Callot, Rec., 1880, 336.—Delage, J. du Midi, 1838, 67.—Franze, S. B., 1862, 101.—Stoll, W. f. Tk., 1901, 280.—Zündel, J. vét., 1858, 444.

### 8. Lupine Disease. Lupinosis.

**Etiology.** In certain districts in Northern Germany where Lupine (*Lupinus flavus*, *cæruleus* and *albus*) is used as fodder, on account of its high percentage of protein, extensive outbreaks of disease sometimes occur among sheep, which are no doubt due to poisoning by these plants. In these districts the lupines are harmful only in certain years and in any year the plants may be poisonous on certain fields only. They are seldom poisonous in the fresh state, but only after a period of storage. Lupine never causes disease when it has been exposed to rain or frost. The yellow lupine is the most dangerous, and the seeds, pods, leaves and straw are equally toxic. Goats, oxen and horses are also attacked. Cases are rare in horses, as they do not like lupine on account of its bitter taste.

The investigations of Arnold & Lemke, Liebscher, Kühn and Roloff, regarding the poisonous substance contained in lupine are well known. It may be extracted from the plant by an aqueous alkaline solution, such as 2% soda. The extract causes the same symptoms as the plant itself, and the plant is non-toxic after extraction. The toxic substance has received a variety of names. Kühn calls it "Ictrogen," and Arnold and Schneidemühl, "Lupinotoxin." It is with difficulty soluble in water, insoluble in glycerine, alcohol and ether. It is not destroyed by a temperature of 190° C. dry heat even after three hours but by steam at 2½ atmospheres pressure its pathogenic power is lost.

The circumstances which control the production of lupinotoxin are not yet known, but it cannot be denied positively that the toxin is produced by fungi which grow on dead plants or on the food itself after storage, and that it is allied to the bacterial and fungoid toxins. This view is supported by the facts that the toxicity is retained in food that



has been left in the open for a long time, and also it would be quite impossible to explain why lupine grown on neighboring land fed at different times and in different manners sometimes shows great variation in toxicity.

Arnold and Schneidemühl's method of extracting the toxin: Finely ground lupine is mixed with 1½% soda solution to produce a thin pulp. This is maintained at 40 to 50° C. for two days and then subjected to pressure. The liquid so obtained is placed on a water bath at 60° C. and after the addition of acetic acid is filtered. Lead acetate is then added and sulphuretted hydrogen passed. It is then evaporated at 50° C. and the semi-liquid residue is mixed with 15 volumes of alcohol. The precipitate which falls in about 24 hours forms when dried a brown shining resinous mass. Five grams of this produce typical symptoms in a dog. After further purification the material is found to be of an albuminous nature.

Liebscher has isolated and experimentally investigated the alkaloids contained in lupine and has shown that they always produce paralysis of the medulla oblongata, and further, that absorption from the stomach is very difficult and that no causal connection can be established between them and lupinosis.

**Pathogenesis.** Lupinotoxin principally affects the mucous membrane of the alimentary canal and after absorption affects the liver, setting up fatty degeneration and catarrh of the gall bladder. The toxin circulating in the blood causes parenchymatous degeneration and fatty degeneration of all the solid organs.

**Anatomical Changes.** In very acute cases there is, in the early stages, cloudy swelling of the liver and jaundice, either of the liver only or of the whole body. Later, fatty degeneration occurs, the liver appearing soft, easily torn and of an intense yellow color. There may be a number of red foci. There is a simultaneous parenchymatous degeneration or fatty degeneration of the kidneys, heart muscle, and certain groups of muscles, while there is severe inflammation associated with small hemorrhages in the abomasum, ileum and large intestine. There are numerous hemorrhages in the serous membranes, skin and subcutaneous connective tissue, and also an edematous infiltration.

In chronic poisoning the liver shows chronic interstitial inflammation, and not rarely a nodular condition produced by shrinkage. These lesions are associated with the presence of fluids in the body cavities, enlargement of the spleen and chronic nephritis.

**Symptoms.** In sheep the first symptom is lack of appetite, especially for lupine, other foods being eaten for some time longer. At almost the same time symptoms of cerebral excitement or great depression occur. There is great weakness. The animals lie down a great deal or stand with their heads hanging, making vague chewing movements, grinding their teeth, swaying from side to side until they drop quite unconscious.

According to Roloff the temperature is elevated at the onset of the disease but it shows pronounced irregularity, and towards the end there is a marked fall. Both pulse and respiration are greatly accelerated in the later stages.



In the majority of cases there is jaundice of the visible mucous membranes, especially in animals that are most severely attacked. At the commencement there is constipation and the dung is hard and covered with yellow mucus. Later there is diarrhea, the feces being mixed with blood. The urine is passed at short intervals, it is dark red in color and contains large quantities of albumin, bile pigments and bile acids. In the sediment there are different kinds of epithelial cells and renal casts.

In occasional cases there is an erysipelatous swelling of the skin of the head, especially in the region of the ears and nose. A serous yellow liquid exudes from the surface which dries up and forms crusts (Stöhr). Exceptionally there is a purulent or sanguinolent discharge from the nose (Roloff).

In the horse the symptoms presented are more or less like those in the sheep, especially with regard to the refusal of food and the intense depression and jaundice, which are the most prominent symptoms. There may also be a yellow mucous discharge from the nose, erysipelatous inflammation of the skin of the nostrils and lips, which may become gangrenous, and inflammatory edema of the limbs.

**Course.** In acute cases death occurs in 3 to 5 days, but in the majority of cases it is delayed until the 9th or 11th day. If the food be changed immediately when the symptoms appear, improvement sets in about the 5th or 6th day, and there is rapid and complete recovery. In a proportion of cases the recovery is not complete and the animals die later, showing symptoms of chronic hepatitis and general cachexia.

If lupines that are toxic to only a slight degree be used for food over a long period, symptoms of chronic lupinosis develop, the animals showing progressive anemia and cachexia. Jaundice is absent or only slight, but inflammation of the skin is sometimes observed.

**Treatment.** In the absence of an antidote efforts must be directed towards preventing the absorption of the toxin by giving acid solutions and removing the toxin from the intestine as rapidly as possible by means of oil purgatives (Roloff).

If the lupine has been proved to be poisonous, its use must be discontinued for a time. The food can be rendered harmless by exposing it to rain in small heaps and using only the superficial layers for feeding purposes, or it may be soaked in 1% soda solution, with renewal of the liquid every two days, and then dried, or by submitting it to steam at a pressure of two atmospheres. Slightly poisonous lupine may be mixed with good food in the proportion of 1:6-10 without danger. Finally it is advisable to test every fresh supply of lupine upon a few sheep.

**Literature.** Roloff, A. f. Tk., 1883, IX, 1.—Schneidemühl, Vortr. f. Tzta., 1883, VI, H, 4 (Lit.).

## 9. Suppurative Hepatitis. *Hepatitis suppurativa.*

(*Embolic hepatitis.*)

**Etiology.** Suppurative hepatitis is usually a metastatic lesion. The pyogenic bacteria, responsible for its production (streptococci and staphylococci), reach the liver either by way of the arterial blood or by way of the portal vein. In cases where the infection occurs by way of the portal vein, the source of the infection is gastro-enteritis, and especially when there is ulceration or necrosis of the mucous membrane (enterogenous abscess of the liver [Joest]). Other causes are suppuration of the spleen, intestinal wall or mesenteric glands and, finally, disintegration of thrombi in the umbilical vein in new-born animals. According to Schumann, the abscesses in the livers of calves are associated with suppurative inflammation of the navel (omphalogenous abscess of the liver [Joest]). Pyogenic bacteria may gain access to the liver by way of the blood stream in any severe inflammatory condition, but especially in strangles, ulcerative endocarditis, and gangrenous pneumonia. Infection may occur by way of the bile ducts, owing to necrosis and inflammation of the walls, caused by gallstones, foreign bodies (ears of grain, sand) or animal parasites, the infection then spreading to the liver tissue.

Injuries to the liver are most common in cattle and are usually due to the penetration of foreign bodies from the stomach. Injuries from the outside, such as horning, may have the same results. Pyogenic bacteria may be introduced into the liver in this way or the liver tissue may be broken down, and any bacteria circulating in the blood may localize there.

Finally, suppuration is frequently caused by echinococcus cysts, tuberculosis and actinomycosis. In these cases the primary condition is of special importance. In nodular necrosis of the liver there is sometimes liquefaction of the necrosed tissue, leading to the production of abscess-like lesions, which differ from the true liver abscesses in that they contain only structureless débris.

At the Budapest slaughter house during a period of 5 years suppurative hepatitis was found in 0.2% of slaughtered cattle and in 0.09% of calves. Lisi found numerous abscesses in the livers of lambs.

In the tropics abscesses in the liver are of very common occurrence and are very often associated with enteritis. The disease has been observed by Smith in India and by Griffault in the Sudan.

**Anatomical Changes.** The liver contains numerous abscesses, varying in size up to a nut. Their contents may be creamy or dry and crumbling. The color varies from yellow or reddish to green. Occasionally the pus has an offensive odor. In suppurative hepatitis of umbilical origin in calves, the abscesses are principally found in the left lobe, on account of the

distribution of the portal vein (Schumann). In recent cases the liver tissue is in a condition of parenchymatous or fatty degeneration, but in cases of longer standing the abscesses are provided with walls of fibrous tissue and the surrounding liver tissue is traversed by strands of fibrous tissue. In other words, the lesion is one of chronic interstitial hepatitis. The superficial abscesses project above the level of the liver tissue and fluctuation can be felt. The serous membrane of the liver may be covered with fibrin or thickened.

For various reasons the abscesses are usually few in number, and when due to an injury there is, as a rule, only one. In such cases the abscesses may attain an enormous size. Felisch reports one in which the abscess measured 20 cm. in its greatest diameter. Abscesses of this size project above the surface of the liver and are usually adherent to the diaphragm, or possibly to the stomach or large intestine. In rare cases rupture of such an abscess causes a suddenly fatal peritonitis.

In the liver abscesses of an ox Grips found the bacillus pyogenes bovis. Künemann repeatedly found the necrosis bacillus and Lisi found staphylococci in the abscesses in the livers of lambs. According to Schumann omphalogenous abscesses in calves are caused by various bacteria but especially by streptococci and staphylococci. In other cases he found colon bacilli and the bacillus pyocyaneus. The abscess-like necrotic foci are caused by the necrosis bacillus. (Bürgi has described a purulent hepatitis of the hare that is due to a staphylococcus.)

According to Stubbe an abscess on the anterior surface of the liver sometimes elevates Glisson's capsule and becomes adherent to the diaphragm, the extent of the adhesion gradually extending until the abscess wall loses its connection with the liver and remains attached to the diaphragm, the convexity of the liver showing a depression corresponding to the position of the abscess.

**Symptoms.** Single and, occasionally, numerous small abscesses may produce no noticeable symptoms, or at most, a certain amount of wasting. In other cases suppurative hepatitis is a complication of another disease, and thus the symptoms to which it gives rise are completely obscured. It is only in rare cases that a moderately rapid process of abscess formation in the liver causes visible symptoms. In addition to disturbances of digestion, there may be persistent fever of no particular type, or there may be simply occasional elevations of temperature. An increase in the amount of indican in the urine is demonstrable. These symptoms are indicative of some internal suppurative process; the more or less pronounced jaundice, and the presence of bile pigments in the urine point to suppurative hepatitis (Smith).

In the dog and ruminant enlargement of the area of hepatic dulness has some diagnostic importance, as has also swelling in the hypochondriac region in the former. In all animals there is pain on pressure over the hepatic area. According to Moulleron and Chauffart, hepatic dulness can be discovered in the horse in some cases. In cattle there may be symptoms of traumatic gastritis (indigestion, shallow respiration and groaning



during movement). According to Griffault, the following symptoms may be seen in the horse: Susceptibility to pressure over the hepatic area, pain during defecation, coughing and straining, marked fullness of the veins of the abdominal wall, edematous swellings and irregular temperature. The enlarged liver may be palpated on the right side behind the costal arch. Albrecht observed coughing in the horse.

In order to ascertain the position of the liver-abscess Griffault advises puncture in the 16th or 17th intercostal space close under the costal arch. The operation is not without danger as infection of the peritoneum is likely to occur if the abscess is superficially placed.

**Treatment.** Internal treatment is utterly useless. If the diagnosis be sufficiently certain, surgical intervention may be attempted.

Griffault operated as follows: An incision was made through the abdominal wall as far as the peritoneum, immediately below the costal arch on the right side. The peritoneum was then approached to Glisson's capsule so that its inner surface was in contact with the abscess wall. The abscess cavity was drained and washed out and a bandage applied. In this way two animals were cured.

**Literature.** Albrecht, W. f. Tk., 1898, 1. Bürgi, Cbl. f. Bakt., XXXIX, 549; 1906, XL, 79.—Griffault, Bull., 1904, 81, 402.—Künneemann, A. f. Tk., 1903, XXIX, 128.—Lisi, N. Ercol., 1907, 49.—Moulleron & Chauffart, Rec., 1906, 25.—Schumann, Unters. üb. Abszesse in d. Leber, d. Kalbes. Diss. Leipzig, 1908 (Lit.).

## 10. Nodular Necrosis of the Liver. *Necrosis nodosa multiplex hepatis.*

Nodular necrosis of the liver is characterized by the presence of tumor-like, dry nodules in the liver substance, which undergo a process of gradual softening from the periphery.

**Occurrence.** The disease is of very frequent occurrence in cattle. Cases in the sheep are more rare, and in the horse, pig and dog quite exceptional. In the sheep it sometimes occurs as an enzootic (Berndt).

**Etiology.** As shown by the investigations of McFadyean, Bang, Schütz, Kitt and Meyer, the cause of the condition is the necrosis bacillus (Bang).

In natural cases the liver becomes infected by way of the blood stream. In most cases the bacillus reaches the portal blood from the intestine. It is not essential that any lesion should be produced in the mucous membrane. In new-born calves infection may take place by way of the umbilical vein. In 15 cows examined by Berndt, either just before or just after calving, the bacillus could be found in the diseased uterus. In certain cases a connection can be established between the disease and the use of very dusty or mouldy food, or the stabling of the animals in dirty stables.

Eisenmann found necrotic foci in the liver associated with chronic inflammation in some cases of swine erysipelas. Caseous foci are sometimes found in the liver of sheep and calves due to pseudo-tubercle bacilli. These foci bear considerable resemblance to necrotic lesions in the process of softening.

**Pathogenesis.** The necrosis bacillus is generally arrested in the smallest branches of the portal vein, or occasionally in the hepatic artery. Multiplication takes place there and the bacilli pass through the vessel wall and penetrate into the surrounding liver tissue. The center of the lesion gradually becomes necrotic and the bacilli are to be found in the peripheral parts arranged in radiating bundles of filaments. An inflammatory reaction sets in around the necrotic area, produced in part by the products of tissue destruction and bacterial toxin and in part by the necrotic tissue acting as a foreign body (McFadyean).

**Anatomical Changes.** The liver, which may be more or less enlarged, is either normal or slightly yellowish in color. The surface of the organ shows sharply defined rounded protuberances of a light brown or yellow color. These are of a firmer consistency than the surrounding tissue. The capsule of the liver covering the superficially placed nodules is thickened and covered with a thick layer of fibrin. Some of the superficial nodules may be eroded and covered with a purulent liquid. There is often secondary sero-fibrinous peritonitis, which is most marked in the neighborhood of the liver.

When the disease has been in existence for a long period the nodules comprise a dry necrotic center, a zone in which the inflammatory reaction has produced softening and an external capsule of fibrous tissue. Finally the central part becomes converted into a viscous yellow material resembling pus, and is surrounded by a fibrous capsule which may measure 3 mm. in thickness.

In cases of chronic swine erysipelas Eisenmann found the liver reduced in size and beset with white or yellowish centers.

**Symptoms.** According to Berndt the symptoms are inappetence, high fever, weakness and painfulness of the liver. The animals move with difficulty, respiration is accelerated and shallow, pressure over the liver causes pain. After three days the animals become very weak and lie down continuously. Respiration is very rapid and difficult, there is constipation followed by diarrhea, the abdomen is painful, the temperature falls, jaundice sets in and the animals soon die. Otto saw two cases in which there were symptoms resembling those of parturient paralysis after the disease had been in existence for 1 to 1½ weeks.

In cases where the number of lesions is small, or where subsequent processes are less rapid, the symptoms are not severe or there may be no evidence of any disturbance of health.



**Diagnosis.** If no suppurative process can be discovered in any other part of the body and if the cow be in the last stages of pregnancy, or just calved, the symptoms already described (pain on pressure over the region of the liver, jaundice, and later evidence of peritonitis), justify a suspicion that the disease is in existence. It is, however, difficult to arrive at a certain diagnosis or to exclude the possibility of purulent or pseudotuberculous hepatitis.

**Treatment.** Treatment has so far proved useless and the immediate slaughter of the animal is indicated.

**Literature.** Berndt, A. f. Tk., 1895, XXI, 194; Pr. Vb., 1903, II, 35.—Eisenmann, Monh., 1907, XVII, 97.—McFadyean, J. of comp. Path., 1891, 46.—Meyer, Unters. üb. d. multiple Nekrose d. Leber d. Rindes, Inaug. Diss. 1903 (Lit.).—Otto, S. B., 1899, 88.—Storch, Pr. Vb., 1903, 36.

### 11. Chronic Interstitial Hepatitis. *Hepatitis interstitialis diffusa chronica.*

(*Cirrhosis hepatis.*)

This condition consists essentially in a proliferation of the interstitial tissue at the expense of the liver substance. In some cases the new fibrous tissue does not contract, either for a long time or at all, and atrophy of the liver substance is seen only at places (*hepatitis indurativa hyperplastica*). In other cases there is an early shrinkage of the connective tissue and a consequent reduction in size of the liver and destruction of liver parenchyma (*cirrhosis atrophica hepatis*).

**Occurrence.** In certain districts the disease is of frequent occurrence and not rarely there are outbreaks, causing great losses, since, sooner or later it has a fatal termination. Large numbers of cases are observed under certain conditions. Besides these, large numbers of cases are due to the migrations of animal parasites, especially in pigs, sheep and oxen. Otherwise, the disease is sporadic in all animals.

The form of chronic hepatitis known as "Schweinsberg Disease" was first observed in Schweinsberg, Ohmtal. Since then it has been seen in some districts in Bavaria and the Rhine provinces. The disease varies in severity from year to year and attacks freshly imported horses more frequently than native ones. In South Africa the disease occurs among horses and cattle, both sporadically and as an epizootic (Robertson). In New Zealand it is known under the name "Winton's Disease" (Gilruth), in Nova Scotia as "Pietou Cattle Disease" (Wyath-Johnston), and in South Dakota as "Bottom Disease" (Schröder, Smith).

**Etiology.** There is no doubt that the long continued administration of poisonous materials produces primary chronic hepatitis. In this connection plants used for food call for special consideration. These plants in swampy districts contain some poisonous material, the nature of which is not yet known. The



so-called "Schweinsberg Disease" was seen only in horses that had grazed on lands which are often flooded, or that had received food grown on such land, while the farms on higher ground in the same district never had cases of the disease or suffered only to a slight extent. The plants grown in these districts contain some substance that is irritating to the liver tissue. Schlegel and Adelman have recently shown that the disease may be no more than a generalized sclerostomiasis, and that the chronic inflammatory changes in the liver are produced by the migrations of the larvæ of sclerostomes.

The idea that certain plants contain substances capable of causing chronic hepatitis does not agree with the experience obtained with regard to chronic lupinosis. However, the disease has been observed after long continued feeding with several plants of the *Senecio* group (*S. Jacobea*, *S. latifolia*, *S. Burchelli*) in New Zealand, North America and South America (Smith, Gilruth, Robertson, Wyath-Johnston, Schröder), and it has been set up experimentally in cattle and horses by Gilruth and Robertson. Guittard has observed chronic hepatitis in geese fattened on maize.

Gilruth fed two six-months-old calves on six pounds of *S. Jacobea* daily. Both calves died at the end of four weeks. To the naked eye their livers appeared unaltered but under the microscope there could be detected a perivascular connective tissue, slight thickening of the capsule and commencing destruction of the liver cells.

Robertson experimented with calves, adult bovines and horses, and found that the administration of large quantities of *S. Burchelli* and *latifolia* caused gastro-enteritis and venous hyperemia of the liver in a few days, while small quantities fed over a period of weeks caused atrophic cirrhosis of the liver.

Since chronic hepatitis is frequently met with in people addicted to alcohol, the idea suggests itself that alcohol may be the cause of the disease in animals, in as much as it is present in the swill tub. One must not lose sight of the effect of fermentation products that may be formed in this food during storage; and especially, as it has been shown by v. Baumgarten and Hansemann to be impossible to produce chronic hepatitis in experimental animals by the systematic introduction of alcohol into their bodies, either by subcutaneous injection or by ingestion.

In certain cases the disease is set up by chemical substances contained in rotten or mouldy food, or by certain digestive disorders of the alimentary canal itself. Of 350 pigs killed, belonging to innkeepers and brewers, Tschauner found 13 affected, but out of 5,700 farm pigs killed at the same time, only 3 were found similarly diseased. The former had been fed on the waste (potato peelings, etc.). Possibly the small percentage of alcohol present among the fermentation products played some part in the production of the disease. In a case described by Begeng the disease was causally connected with a chronic gastro-enteritis. Begeng agrees with Siegenbeck van Henkelom that hypertrophic cirrhosis of the liver is produced by toxic

materials present in the intestine, which are conveyed to the liver by the portal blood and set up irritation, causing proliferation of the connective tissue.

Krawkow was able to cause cirrhosis of the liver in experimental animals with broth made from putrid horse flesh. Boix had positive results with the subcutaneous inoculations of monobasic fatty acids which tend to be formed by abnormal fermentative processes in the intestine.

Disease viruses are probably common causes of chronic hepatitis. Joest produced a condition resembling the Schweinsberg disease, both clinically and as regards the lesions by long continued injections of the bacillus *suisepitici*. Pronounced lesions of chronic hepatitis were observed by Eisenmann in several cases of swine erysipelas, and similar lesions may be sequels to infectious diseases (post-infectious chronic hepatitis).

Langer found in calves' livers numerous necrotic foci often associated with cellular infiltration, which closely resembled those seen in the human subject in typhoid, cholera, scarlatina and measles. From these nodules a bacillus of the typhoid type was cultivated (*bacillus nodulifaciens bovis*), the cultures of which produced similar nodules in the livers and kidneys of mice, guinea pigs and a calf.

Dantschakowa produced chronic induration of the liver in rabbits by repeated subcutaneous inoculations of the staphylococcus *pyogenes aureus* at intervals of 4 days in 7 to 15 weeks. This was followed by a localized small-celled infiltration and the development of a collagenic tissue in the center of the lesions and peripheral extension of the infiltration.

Finally certain infectious diseases and poisons are capable of setting up chronic cirrhosis of the liver.

The disease is very frequently seen as a secondary condition. It is caused principally by animal parasites (fluke, *Cysticercus tenuicollis*, and the larvæ of sclerostomes). The sheep and pig are affected chiefly, but it is also met with in calves, horses and rabbits. As already mentioned, Schlegel and Adelmann look upon the Schweinsberg disease as a generalized sclerostomiasis (see page 530). The formation of fibrous tissue is due partly to the destruction of liver tissue and partly to toxic materials elaborated by the parasites themselves, particularly the fluke.

Chronic hepatitis may be caused through chronic inflammation of the walls of the bile ducts arresting the flow of bile, and by extension of the inflammatory process from the inter- and intra-lobular bile ducts to the interstitial tissue, the amount of connective tissue being increased. Engorgement with bile may lead to impairment of the nutrition of the epithelium of the bile ducts, and this may be followed by a bacterial invasion and so cause a production of fibrous tissue in the surrounding liver tissue, or the tissue production may be due to the ill-effects of the biliary engorgement on the liver cells themselves.

Purulent foci and tuberculous lesions may cause not only cirrhosis in their immediate neighborhood, but a diffuse lesion throughout the liver.

Chronic venous congestion caused by certain diseases of the

heart and lungs, or by pressure on the posterior vena cava, may cause not only dilatation of the hepatic vessels (nutmeg liver) and an atrophy of the liver cells, but also an increase in the amount of connective tissue. Chronic hepatitis may also be seen in cases of thrombosis of the portal vein, which may either be due to the derangement of the liver parenchyma or may be the result of the cirrhosis, which has some previous cause.

Bleichröder considers that the chronic hepatitis seen in the human subject as a sequel to diseases of the blood originating in the portal area is produced in the following manner. The blood in the portal vein has poured into it an enormous number of lymphocytes from the spleen, these are deposited in the liver and lead to the production of new connective tissue. This process would explain the enlargement of the spleen which is suggestive of an infectious disease. The jaundice is due to extensive destruction of red blood corpuscles and must therefore be considered as a pleiochromic icterus. The so-called Banti's disease of the human subject, the symptoms of which are anemia, enlargement of the spleen, ascites, increased urobilin-content of the urine and frequently cirrhosis of the liver, is also possibly brought about in this way.

**Pathogenesis.** When cirrhosis of the liver is due to some irritant circulating in the portal blood, the process starts in the interlobular branches. There is a cellular infiltration followed by the formation of connective tissue. In this way the amount of connective tissue surrounding several lobules becomes increased in amount. By the subsequent shrinking of this tissue the included lobules and the branches of the portal vein ramifying in the proliferating tissue are subjected to pressure. Degeneration and sometimes necrosis and destruction of the liver tissue follows, partly owing to the constant pressure exerted by the contracting tissue and partly owing to the obstruction of the larger vessels. In the later stages the connective tissue penetrates into the interior of the lobules.

Irritants in the bile ducts lead to the production of the inflammatory changes in their immediate neighborhood (cholangioitic cirrhosis). As shown by Jäger's extensive investigations regarding cirrhosis due to flukes (q. v.), the inflammatory process starts in the angle between adjacent lobules, either as a cholangioitis or as a cellular infiltration of the connective tissue. The process extends along the interlobular septa, following the lymph stream, and finally attacks the peripheral parts of the lobules. Localized cirrhosis appears to be caused in this way. In some cases contraction of the connective tissue is delayed or does not occur at all, but even in these cases there is compression of the interlobular blood vessels.

In Schweinsberg disease of the horse, the inflammation involves the liver parenchyma and especially the blood vessels, producing a perilobular hypertrophic cirrhosis (Mugler). Kitt formerly described the disease as a chronic parenchymatous hepatitis, with secondary formation of connective tissue.

In chronic venous congestion of the liver, the formation of connective tissue commences around the hepatic and central veins and then extends to the interlobular spaces.



If the contraction of the connective tissue involves a large number of capillaries and other vessels, the transverse area of the blood path is greatly diminished and there is consequently congestion in the portal area. Compression or obstruction of the bile ducts, especially in certain forms of cholangioitic cirrhosis, leads to biliary congestion. The destruction of the liver cells affects not only the secretion of bile and the metabolism of the body, but may also induce symptoms of poisoning. In many cases new bile ducts are formed by a process of budding from preexisting ducts.

**Anatomical Changes.** In cases where the connective tissue has contracted, the liver has a tough and, in the later stages, a leather-like consistency. When cut into the tissue grates under the knife. At the outset there may be no visible reduction in the size of the organ and, in fact, it may be a little enlarged. In the later stages the reduction becomes more and more marked, until the liver may be only one half the normal weight. The surface appears irregular and granular. In some cases the surface is nodular. The serous covering may be thickened at places and the edges may be formed simply of a double fold of peritoneum. On the cut surface the compressed lobules stand out like small granules against the pale red or grayish-white connective tissue, which forms a wide network around either groups of lobules or individual lobules. These granules appear of a faint or deep yellow color, with a tinge of green, on account of the fat and pigments contained in the liver cells. At a distance the whole organ looks yellow.

According to Kitt the liver in cases of Schweinsberg disease is at first enlarged, its surface uneven and of a reddish-brown or gray color resembling porphyry or granite. With the development and shrinking of the connective tissue the condition known as "granular atrophy" is produced.

In other forms of chronic interstitial hepatitis the liver appears enlarged. The outer surface and the cut surface appear smooth, or at most somewhat granular and deep yellow or greenish-yellow in color (cirrhosis hypertrophica s. hyperplastica). Livers so affected may weigh as much as 20 kilos in the horse and 20-25 kilos in the ox. Adam observed one case in an ox in which the liver weighed 15.0 kilos (Kitt). In the dog cirrhosis of the liver is frequently associated with fatty degeneration.

In calves still another form of hepatitis is seen, characterized by a diffuse production of connective tissue and by degeneration of the parenchyma. In such cases the liver is tough and firm, yellowish-red or flesh-like in color and covered with whitish spots and streaks. There is acute swelling of the neighboring lymphatic glands.

Rauscher recognized the following types of chronic hepatitis:

1. Diffuse induration, including the so-called "porphyry liver," the enlarged, leaden colored livers seen in cases of distomatosis, and the nutmeg and cirrhotic livers seen in Schweinsberg disease.

2. Nodular induration with irregularities of the surface as large as hens' eggs (hobnail liver of the ox and lobulated liver of the pig).
3. Granular atrophy, seen in the dog, ox and pig.
4. Cicatrized liver, seen in cattle and sheep and due to the cicatrization of burrows in the liver tissue made by parasites (*Cysticercus tenuicollis* and distomes).
5. Nodular fatty liver (dog), characterized by the formation of nodules as large as hazelnuts or larger, and fatty degeneration and jaundice of the liver.

Evidence of congestion of the portal area and jaundice complete the list of lesions. In Schweinsberg disease, pronounced catarrhal gastritis is a constant lesion.

**Symptoms.** The onset of the disease is unnoticed as a rule and even in later stages the symptoms are frequently only those of an indefinite digestive disorder. Capricious appetite, gaping and vomiting (observed by Moens in the horse) indicate severe gastric catarrh. There may be either constipation or



Fig. 67. Enlargement of the area of hepatic dulness in chronic hepatitis in the horse. The area enclosed by the line indicates the area of dulness, the white dots the costal arch, and the numbers indicate the position of the corresponding ribs. The liver in this case weighed 19.7 kilos.

diarrhea. Horses show symptoms of colic after eating large quantities of coarse food (especially straw mixed with dung) causing dilatation of the stomach (Imminger). In cases in cattle, caused by plants of the genus *Senecio*, there is severe diarrhea, tenesmus and often in consequence prolapse of the rectum. Horses show symptoms of "sleepy staggers." Persistence of these symptoms causes the animal to lose condition. The mucous membranes appear pale and sometimes yellow. In Schweinsberg disease there may be early congestion of the mucous membranes. The skin is dry and its elasticity is diminished. The coat is rough and there is progressive wasting. Animals lose their energy, tire easily and show signs of broken wind.

The enlargement of the liver is only exceptionally sufficient to cause it to encroach upon the hypochondriac region. In the



horse, carnivora and swine, this encroachment may be bilateral but in the ox it is only on the right side and then only as far as the anterior part of the hollow of the flank. Similarly it is only very exceptionally that the enlargement is sufficient to allow of palpation of the organ per rectum. In these cases it can be felt in the neighborhood of the last rib as a firm object lying against the abdominal wall and moving in concert with the respirations. Far more frequently in cattle the upper border of the enlarged liver can be felt by pressing inwards with the fingers behind the last rib. In carnivora and emaciated swine the liver can be felt through the abdominal wall under both costal arches, in calves and small ruminants under the right costal arch only. The hepatic area is very susceptible to pressure, animals experiencing difficulty and exercising great care in turning.



Fig. 68. Enlargement of the area of hepatic dulness in a cow due to chronic hepatitis. A. Normal area. B. Enlarged area. The dotted line marks the position of the costal arch and the numbers indicate the corresponding ribs. The liver weighed 14.6 kilos.

An increase in the area of hepatic dulness may be referred without any further delay in carnivora, ruminants and swine, to a somewhat pronounced enlargement of the liver. The same holds good for the horse. In carnivora (figs. 69 and 70) the enlargement extends downwards and backwards, usually reaching the umbilical region. In ruminants (fig. 68) it extends backwards and downwards to below the costal arch on the right side, or into the anterior portion of the hollow of the flank. In ruminants the normal area of dulness depends upon the quality of food in the stomachs and intestines. In the horse (fig. 67) in cases where the enlargement of the liver is considerable, there is an area of dulness immediately behind the edge of the



lung on the right side from the tenth to seventeenth intercostal spaces. This may extend downwards to below the costal arch. Under similar circumstances there is a smaller area on the left side below the edge of the lung from the eighth to the tenth intercostal spaces.



Fig. 69. Enlargement of the area of hepatic dulness in a dog due to chronic hepatitis. H. The enlarged area extending to the navel in the forward direction, as far as the cardiac area (C). The costal arch is indicated by the dotted line. The dog was of medium size, but the liver weighed 3 kilos.

Marked reduction in the size of the liver is sometimes indicated by a decrease in the size of the area of hepatic dulness. In these cases in the smaller animals the liver can be felt from the costal arch only, the firm consistency and uneven surface can, however, be appreciated.



Fig. 70. Enlargement of the area of hepatic dulness in a dog due to chronic hepatitis. The right side shown in Fig. 69.

Fairly frequently, and especially in dogs, there is ascites which may persist without complications till death, or towards the end of the disease there may be edematous swelling of the abdominal wall and legs.

Enlargement of the spleen can be diagnosed in carnivora and emaciated pigs by palpation of the abdomen. In the horse it may sometimes be discovered by rectal examination.

In cattle, jaundice is frequently seen, and towards the end it is very severe. In other animals it usually sets in at a late stage and is not very pronounced even then. This is quite in accordance with the fact that the hyperplastic form of the disease is the commonest in cattle.

The specific gravity of the urine is higher, it is darker in color and contains less uric acid than normal; but in some cases albumin, sugar and urobilin are present. (In a liter of urine from a horse, Mouquet found 7.25 gm. of uric acid, 0.12 gm. of albumen and 15.4 gm. of sugar. Urobilin was also present.)

Towards the end of the disease, nervous symptoms make their appearance, the animals becoming dull and lethargic. In Schweinsberg disease and condition caused by plants of the order *Senecio*, these symptoms are sometimes very severe, and appear in the early stages. There is giddiness and unsteadiness of gait, the animals lean forward with their heads against the wall and attempt to go forwards, make gaping movements and seem dull and sleepy. The appearance is suggestive of staggers (Beichold, Imminger, Robertson).

In cases of secondary cirrhosis of the liver the symptoms of the primary disease are present.

**Course.** The disease lasts for several months (usually from three to six months in the case of Schweinsberg disease). The disease of the liver and the subsequent catarrh of the stomach and intestine cause a loss of condition, pronounced wasting and anemia. In the later stages there are often dropsical swellings. Finally in the last stages there may be hemorrhages in the mucous membranes, gums, skin, stomach or intestines. Exceptionally rupture of the liver or dilatation of the stomach (in the horse) may cause death unexpectedly, death usually occurring only after the animal has reached a state of utter exhaustion.

**Diagnosis.** The symptoms are never easily interpreted. The insidious development of the disease, persistent digestive disturbance, the possible presence of ascites and enlargement of the spleen and sometimes of jaundice render diagnosis a possibility, but do not absolutely exclude the chance of error. Digestive disturbances of this nature in horses in districts where it exists suggest Schweinsberg disease. The following diseased conditions of the liver cause a similar train of symptoms: carcinoma of the liver in dogs (large tumorlike growths in the liver are particularly suggestive of this), amyloid liver in which the organ is enlarged and firm, but its edges rounded and smooth, peritonitis (the abdomen sensitive to pressure and

sometimes elevation of temperature), chronic heart disease (cardiac sounds and evidences of congestion in various parts of the body). In cases of ascites paracentesis abdominis should be practiced, thus rendering thorough examination of the liver more easy through the relaxation of the abdominal wall.

It is rarely possible to distinguish with certainty between hyperplastic and atrophic cirrhosis of the liver. Apart from the changes of shape that may be ascertained by palpation and the enlargement of the area of hepatic dulness, it may be taken that ascites and enlargement of the spleen indicate atrophic cirrhosis, while pronounced jaundice suggests hyperplastic cirrhosis.

**Prognosis.** The disease is inevitably fatal, but there may be temporary improvements.

**Treatment.** In the first place the probable cause should be removed. The diet should receive careful attention, all sour grasses or irritating materials should be excluded, and a change of food should be given. Neutral salts may be tried, and the ascites may be combated by the administration of diuretics and by repeated tapping. Imminger advises intratracheal injections of Lugol's solution in cases in the horse; others have had no good results from this treatment.

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## 12. Neoplasms of the Liver. Neoplasmata hepatis.

### Cancer of the Liver. Carcinoma hepatis.

**Occurrence.** Cancer of the liver is rare in all the domestic animals but among them it is most frequently seen in the dog.

**Anatomical Changes.** Primary cancer of the liver is usually adeno-carcinomatous in type. Secondary cancer of the liver generally follows primary growths in the stomach and intestines, in which cases the cancer cells are introduced in the portal blood. The primary growths may also be situated in the peritoneum, mammary gland, pancreas, etc. The lesions in the liver may be



er. They are either white or pale red in color. In many cases they can be enucleated. The superficially placed growths may show a slight central depression (Krebsdelle). The cut surface shows a sulphur- or butter-yellow network. The consistency varies from that of marrow to that of fibrous tissue. Lesions in the livers of cattle are frequently soft, and contain a semi-fluid pulplike material, and in color they scarcely differ from the normal liver tissue (Kitt). The portal lymphatic glands are almost always affected. In cases of primary hepatic cancer, there is usually only a single large tumor, but metastatic lesions are almost always numerous. The weight of the organ is increased according to the number and size of the lesions. Chauvrat records a horse's liver weighing 21.6 kilos; van Tricht, the liver of a cow weighing 22 kilos, and Wilhelmi, one weighing 51 kilos.

Cases of cancer of the gall bladder have been observed (Gurlt, Bruckmüller, Kitt, Johne). The gall bladder is enlarged and nodular, the wall is thick and firm and the inner surface shows either pedunculated or sessile growths which at places are ulcerated.

**Symptoms.** Symptoms are first observed when the disease has made some progress. There is gradual wasting and anemia, the appetite is diminished, and in some cases there is vomiting. In the dog there is striking atrophy of the muscular system, the muscles of mastication being chiefly affected. This gives the animal a peculiar facial expression ("Krebsgesicht," "Facies cancéreux" [Trasbot]).

In ruminants and dogs an enlargement of the area of hepatic dulness can sometimes be demonstrated, or in small animals the enlarged liver may be felt below the costal arch. If small nodules can be discovered, the suspicion is at once aroused that the case may be one of cancer of the liver, especially if other lesions (such as carcinoma of the mammary gland) suggest that possibility. It must always be borne in mind that tuberculous lesions in the liver may cause a similar clinical condition and that the tuberculin test may be negative.

Nodules or growths may also be felt just behind the sternum in cases of enlarged portal or mesenteric lymphatic glands, tumors in the pyloric region, pancreas, or mesentery. Finally greatly distended gall bladders and bile ducts must be kept in mind.

In some cases there is jaundice, and in others ascites and swelling of the spleen.

**Course.** Several months may elapse after the appearance of the first symptoms, but finally the gradually increasing cachexia leads to complete exhaustion.

**Treatment.** Spontaneous recovery never occurs and internal treatment is useless. No very satisfactory results are to

be expected of surgical interference. (Parascandolo removed a carcinoma from the liver of a sheep with good results.

**Literature.** Kasperek, T. Z., 1907, 470.—Markus, Beitr. z. path. Anat. der Leber etc. Diss. Bern, 1902 (Lit.).—Parascandolo, Clin. Vet., 1901, 598.—Wilhelmi, Schw. A., 1903, XLV, 156.

**Other Neoplasms in the Liver.** From a clinical point of view these have only a subordinate interest as they generally remain unobserved during the life of the animal and can only be distinguished from carcinomata when the primary growth is situated in some part of the body or in some organ that is more accessible to investigation. Sarcoma of the liver most closely resembles carcinoma of that organ. Primary sarcoma of the liver is extremely rare, but metastases from growths in the peritoneum, intestine or mesentery are observed somewhat more frequently. In these cases also, there is a gradually progressive cachexia, enlargement and sensitiveness of the liver, and in some instances jaundice and ascites. Cadéac records a case in which the neoplasm involved the wall of the portal vein in an ox and caused the animal's death. In the liver of the horse melanomata and melanotic sarcomata are not absolutely rare and these may be present in large numbers.

Adenomata (Ad. simplex, fibrosum, Cholangiosum viride) are very rare and cause symptoms only when they become carcinomatous.

Angiomata are of fairly common occurrence in the ox, but rarely cause any symptoms. The lesions are generally numerous. (Hemangioma cavernosum or Telangiectasia capillaris.) Trasbot records a case in which a horse which had shown slight symptoms of colic a year previously, died from hemorrhage following rupture of an angioma, and Ball saw a similar case in a cat.

Trasbot found two lipomata in the liver of a dog, one of which was as large as a child's head, and Ratz has seen lipomata in the livers of birds.

Villus-like outgrowths sometimes occur in the gall-bladder, especially in cattle. These sometimes are simple papillomata and sometimes have the structure of a villous cancer. In the latter case, secondary cancer nodules may be present in the liver.

**Literature.** Ball, J. Vét., 1904, 191.—Jäger, A. f. Tk., 1907, XXXIII, 71.—Markus, Beitr. z. path. Anatomie d. Leber. Diss. Bern, 1902.—Rühmekorf, Über multiple dissem. Kapillarekt. d. Leber usw., Diss. Leipzig, 1907 (Lit.).—Ruppert, A. f. Tk., 1909, XXXV, 150.—Saake, D. Z. f. Tm., 1896, XXII, 142.—Trasbot, A. d'Alf., 1879, 241.

**Tuberculosis and Actinomycosis of the Liver.** Tuberculosis of the liver is common in cattle, pigs and birds, more rare in the dog, and is exceptional in the other domestic animals. It is usually secondary to disease of the peritoneum, intestine or mesenteric glands. Occasionally it is embolic and it is also primary in young animals that have been infected by way of the placenta. The liver contains at first only quite small lesions and later caseous or caseo-purulent lesions of considerable size. The material contained in the lesions sometimes resembles mortar. In some cases the lesions have the appearance



of lympho-sarcomata, and very often there is a marked increase in the weight of the organ. In the dog, the lesions sometimes have a central depression, and thus to some extent resemble cancer growths. In sucking animals, it is more or less likely that the disease will only be recognized if, in addition to tuberculous lesions in other organs, enlargement of the liver and a nodular condition of that organ can be discovered.

The lesions of actinomycosis are similar except that they consist of a fibrous capsule enclosing a yellowish, soft, gelatinous tissue which is becoming purulent at places, and in which the actinomyces can be found by microscopic examination.

### 13. Animal Parasites of the Liver.

#### Echinococci.

**Occurrence.** In cattle and pigs echinococci are of very frequent occurrence but rare in solipeds and carnivora. The parasite is also found in man and in turkeys.

The distribution of echinococcosis is intimately related to the number of dogs and the frequency with which they are hosts of the *tania echinococcus*. In Ireland and, according to Vidal, in Tunis also there is scarcely an adult ox or sheep that does not harbor echinococci. The disease is also widespread in Australia and India (70% of all bovines). In Europe, Mecklenburg appears to be affected most seriously. Madelung and Sahlmann found 25 to 50% of cattle, 75% of sheep and 5 to 8% of pigs infested. Metelmann found echinococci in 25% of cattle, 15% of sheep and 5% of pigs.

According to the statistics of 52 German slaughter houses drawn up by Peiper 11% of cattle, 10% of sheep and 6.5% of pigs were affected. In 1896-97 at the Berlin abattoirs the livers of 1,156 cattle, 1,939 sheep and 5,398 pigs were seized out of a total number of 146,612 cattle, 395,769 sheep and 694,170 pigs (Ostertag). According to Längrich the percentages of animals found to be infested at the abattoirs at Rostock were 36% cattle, 26.5% sheep and 5% pigs. In goats and horses the number was 1%. At Stettin, Olt found 7.1% of cattle, 25.8% of sheep and 7.3% of pigs affected. Schmidt calculates the annual loss in cattle to amount to \$37,500. At Budapest during the period 1899-1903 out of 511,031 cattle killed (including 20,000 buffaloes) there were 7,622 cases or 1.4%; 3,755 cases were found among 90,883 sheep (4.1%), and 5,105 out of 474,401 pigs or 1.1%. At Prague the percentage of affected cattle amounted to 23.2% and of sheep 5.5% (Prettner). In Russia the incidence of the parasite varies from 0.1 to 80% in cattle, 0.01 to 60% in sheep, from 0.01 to 70% in pigs and from 0.005 to 40% in horses.

Lichtenfeld's investigations regarding the distribution of the parasite in the various organs are very interesting. These showed that the lungs were affected in cattle in 69.3% of cases, the liver in 27%, the spleen 2.2%, heart 0.75% and kidneys in 0.75%. In the sheep the figures were: lungs 52.2%, liver 44.9%, spleen 2.9%. In the pig the figures were: lungs 18.8%, liver 73.1%, spleen 2.95%, and heart and kidneys in 2.25%. In 1.3% of cases there were parasites in the subperitoneal tissue. In the horse the lungs were involved in 5.5%, and the liver in 94.5% of cases. It was also shown that in pigs under two years old the proportion between the number of cases in which the lungs were affected and those in which the liver was affected is 12.8:82. In adult animals the proportion was 39.3:46.4.

Echinococcus disease is comparatively frequent in man. In the central parts of Europe 1 case is seen in 130 postmortems. *Echinococcus multilocularis* is practically confined to places where cattle are principally affected (Tyrol, Inntal and the Memmingen district in Bavaria), whereas *E. polymorphus* is seen in those places where sheep are the principal hosts (Iceland, Australia, Mecklenburg, Pomerania, Dalmatia, Argentine).



**Etiology.** Echinococci develop from the embryos of the *tænia echinococcus* (Siebold) which inhabits the intestine of the dog. These embryos are liberated from ingested eggs in the stomach and pass out of the intestine into the portal circulation. The vehicles of infection are food or water contaminated with the feces of dogs or with proglottides or tape-worm eggs contained in these.

Echinococci are cysts of various sizes containing a clear, pale, yellow liquid the reaction of which is either neutral or slightly acid. The liquid contains a little albumen, a considerable proportion of salt, and not infrequently succinic acid. The wall is composed of two layers. The outer or chitinous wall is the thicker, white in color and under the microscope appears to be composed of a number of parallel laminae. The inner or germinal layer is yellowish in color, granular, and sometimes contains in the deeper parts muscle fibres, granules of lime and small blood-vessels. In the livers of cattle and more rarely in the livers of pigs and sheep, a second form of parasite occurs, *E. multilocularis* or *alveolaris*, in which there are masses of cysts about the size of peas united together by a fibrous, net-like matrix derived from the outer layer. In this way tumor-like growths of various sizes are formed. The central part is often caseous or calcified, while the peripheral parts are composed of numbers of small cysts resting upon each other, and the whole growth appears to be traversed in all directions by a recognizable fibrous network. According to Ostertag, Müller, Mangold and Posselt, this cyst represents a stage of special tape-worm, the *Tænia echinococcus multilocularis*, (see page 469).

At a certain stage there develop on the inner surface of the cysts small prominences, the so-called brood capsules. Some of the cysts remain attached to the wall and others are free in the liquid. They contain the new scolices. In other cases very small cysts develop between the layers of the wall. Some of these pass inwards and eventually become free in the liquid and later grow into large bladders. There may be large numbers of these daughter cysts within the mother cyst. These may contain granddaughter cysts (*E. endogenus*, *altricipariens* or *hydatisosus*). More frequently the small cysts pass outwards and separating from the parent cyst undergo further development (*E. exogenus*, *scolicipariens simplex*, *granulosus* or *veterinorum*). Both endogenous and exogenous cysts may later give rise to scolices or daughter cysts.

Lichtenfeld found a larger number of fertile cysts in pigs under two years than in older animals and also that the proportion existing between fertile and sterile cysts was in cattle 24:76, in pigs 80:20, in sheep 92.5:7.5 and in the horse 38.9:61.1.

The experimental researches of Bobroff, v. Alexinsky, Dévé and Ponomareff have shown that echinococci may be transported not only to organs in close connection with that primarily infested but also to others, scolices being set free by the rupture of a fertile cyst.

The development of echinococci is very slow. According to Leuckart small foci measuring 0.25 to 0.35 mm. in diameter and enclosed in thin fibrous capsules are found one month after infection. After two months they measure about 2.5 mm. and are already converted into small cysts. Towards the end of the fifth month the cysts are about the size of hazelnuts, the two layers of the wall are well differentiated and the development of the scolices and daughter cysts has begun.

**Anatomical Changes.** The liver is enlarged in proportion to the size and number of the cysts present in it, and in the ox may weigh as much as 158 pounds (Ringk). In the pig, in-

**Symptoms.** In **cattle** digestive disturbances, lasting for weeks or months, have been observed in some cases: capricious appetite, inactivity of rumination, constipation, frequent tympanitis, etc. Exceptionally jaundice and wasting and the general symptoms of ill-health have been seen. The area of dullness may be greatly increased and may extend backwards to the last rib and in the downward direction to the lower third of the abdomen (see fig. 68, page 536). Palpation of this region may cause symptoms of pain. The enlargement of the liver may be so great that the right half of the abdomen and right flank are rendered prominent. By manipulation of the right side of the abdominal wall, and by rectal examination, one can, in cases where the enlargement of the liver is excessive, feel the thickened and rounded edge of the liver and the elastic nature of the prominences can be appreciated (Labarrère, Ländler). Respiration is usually shallow and accelerated. Weinberg and Vieillard found that echinococcosis can generally be diagnosed by the fixation of complement method.

The symptoms in the **sheep** are similar to those described. The disease can only be distinguished from the similar condition caused by flukes if the unevenness of the surface of the liver can be felt through the abdominal wall.

In **pigs** Lucas also saw pronounced ascites in cases of heavy infestation. The animals were quite unable to get onto their hind feet and dragged them behind them. In spite of a good appetite, there was marked wasting. In a case recorded by Schmidt, there was evidence of jaundice, while in one published by Friedrich there was enormous distension of the abdomen without any ascites.

**Treatment and Prophylaxis.** No satisfactory method of treating echinococcosis is known. Since the disease is set up by the ingestion of the eggs of the *Tænia echinococcus*, animals should be prevented from eating food or drinking water that are soiled with the feces of dogs. As this is generally impracticable care must be taken that all organs of animals slaughtered that contain echinococci are destroyed and not, as so often happens, given to the dogs.

**Literature.** Feuerissen, D. t. W., 1908, 110.—Friedrich, B. t. W., 1906, 17.—Criglio, Clin. Vet., 1906, 409.—Joest, Z. f. Infkr., 1907, II, 10.—Labarrère, Rev. Vét., 1887, 619.—Ländler, A. L., 1907, 207.—Lichtenfeld, Cbl. f. Bakt., 1904, XXXVI, 546, 651; XXXVII, 64 (Lit.).—Lucas, Z. f. Flhyg., 1907, XVII, 267.—Martin, Rev. Vét., 1907, 668, 734, 800 (Lit.).—Ostertag, D. Z. f. Tm., 1891, XVII, 172; Fleischschau, 1904, 464.—Pécard, Bull., 1906, 591.—Posselt, Münch. m. W., 1906, 537, 605 (Lit.).—Putzu, Cbl. f. Bakt., 1910, LIV (Orig.), 77.—Schmidt, Z. f. Flhyg., 1907, XVII, 270.—Vidal, Rev. Vét., 1905, 240.

#### (b) Distomatosis. Fluke Disease.

##### (*Liver rot.*)

The disease is caused by the *Distomum hepaticum* and *D. lanceolatum*, and it occurs more frequently in sheep than in cattle. It is an acute or chronic inflammation of the liver and

bile ducts, more commonly the latter. In the chronic form it leads to serious loss of condition.

**Historical.** The earliest records of the disease are those of Schaper von Gabucinus in 1547, and of Gemma a few years later. The term, "Leberegel" (fluke) was first used in 1676 by Frommann. Valuable clinical and pathological studies of the disease were made by Schäffer (1764), Goeze (1782), Chabert (1879), and Billhuber (1791). The life-history of the parasite was elucidated by Mehlis (1831), v. Nordmann (1832), Escherich (1841), Steenstrup (1842), and especially by Leuckart (1876), who determined the metagenesis of the fluke. Among veterinary surgeons the names of Gerlach (1854), Delafond (1854), Davaine (1860), Friedberger (1878), Zündel (1880), Thomas (1881), and Zürn (1882), are closely connected with the study of distomatosis. Interesting investigations were made by Schaper (1889), and Lutz (1892), regarding natural infection, and also by Schaper in connection with the pathogenesis, symptomatology and pathological anatomy of the disease.

**Occurrence.** The *Distomum hepaticum* is found in marshy places everywhere, whereas the distribution of the *D. lanceolatum* appears to be more restricted, the parasite being far more common in southern Europe than in northern. The geographical distribution of the *D. hepaticum* exactly coincides with that of *Limnæus minutus*, which is practically the exclusive intermediate host of the parasite (Leuckart). After wet summers the disease is very widespread and causes very heavy losses. The severe form of the disease rarely occurs among adult cattle, although they may harbor the parasite in their livers. It is principally among calves that serious losses occur. Extensive outbreaks of the disease sometimes occur among goats (Römer). Swine, buffaloes (Hungary and East Indies), camels, wild ruminants, hares and exceptionally horses, asses, dogs, cats and rabbits are infested.

In 1873 a third of the sheep, which were valued at more than \$200,000, died in Alsace-Lorraine. In England the annual loss is computed at a million sheep. Great losses were caused by fluke in Hungary and especially Upper Hungary in 1889. On two farms practically the entire flocks were wiped out. The disease was also very prevalent during the years 1893-1897. According to Popow in certain districts of Poland in 1891 from 50 to 90% of the sheep died. The liver fluke is not known in Iceland (Krabbe).

In 1876 the disease caused losses up to 40% of the cattle in Slavonia.

In 1883-84 36% of all the cattle, 7% of the calves and 1½% of the pigs killed at the Berlin abattoirs were found to be affected. At Budapest during the period 1889-1903 the percentages of affected animals were: 4.9% of cattle, 39.5% of sheep and 25% of goats. In 1902-03, 474,401 pigs were slaughtered and of these 1.2% were seriously infested. At Okayama (Japan) Saito found the *Distomum hepaticum* in 16% of cattle but the lesions were confined to the bile ducts.

**Etiology.** The disease is caused by two varieties of distomes belonging to the Trematodes family of the Platyhelminthes. These two parasites are called *Distomum hepaticum* (*Fasciola hepatica*) and *D. lanceolatum* (*F. lanceolata*, *Dicro-*



*coelium lanceolatum*). Both parasites are leaf-shaped, elongated and unprovided with appendages. The body is broader towards the middle part and there are two suckers at the anterior ends.

*Distomum hepaticum* measures from 18 to 31 mm. in length by 4 to 13 mm. in width, and is covered with minute spines that point backwards. At the anterior extremity there is a conical process which carries at its apex the oral sucker. On the under surface, about 3 mm. behind this, there is a second and larger sucker. The eggs (fig. 72) are brown or greenish-yellow, oval, and provided at one pole with a small cap. This cap may be rendered visible either by exerting pressure on the eggs or by the addition of potassium hydrate solution. The eggs measure 130 to 145  $\mu$  in length and 70 to 90  $\mu$  in width.

The *Distomum lanceolatum* is only 4 to 9 mm. in length and 2.5 mm. broad. It is slender and lancet-shaped. The eggs which are brownish in color are also provided with a polar cap (fig. 71) and measure 37 to 40  $\mu$  in length.

**Life-cycle of the *Distomum Hepaticum*.** According to Weinland, Leuckart, and Thomas, the life-cycle of the parasite is as follows: The eggs of the mature flukes are passed out with the feces of the host into the outer world. Provided segmentation has taken place, an embryo develops under suitable conditions of temperature (at least ten to twelve C.) and moisture in about three to six weeks. The embryo escapes by raising the polar cap. It is elongated in shape and its surface covering is composed of polyhedral cells and thickly covered with cilia. These larvæ (*miracidium*) are very actively motile in water. By means of a movable spine at their anterior end they bore their way into the body of certain fresh-water snails. The snails usually selected are *Limnæus minutus* or *truncatulus*, and more rarely *L. pereger* and others. During the summer months they become converted into sporocysts within two weeks. Each germ-cell gives rise to five to eight rediæ, and each redia to fifteen to twenty cercariæ. The cercariæ pass out of the oral opening of the rediæ, and then leave the body of the snail. The cercaria has a flat oval body measuring 280  $\mu$  in length, and 230  $\mu$  in breadth. It is also provided with a caudal appendage twice that length. Two suckers and a bifid intestine can already be distinguished. After swimming for a time in the water the cercariæ work their way up grass-stalks and there secrete a sticky, mucoid substance which serves both to encapsule them and to cement them to the grass. In this condition the cysts measure about two to three mm. If such cysts be ingested by certain animals the parasites make their way up the bile-ducts and there become sexually mature, according to Leuckart in three weeks, and according to Thomas in five to six weeks. As a rule a proportion of the mature parasites die. The majority, after laying eggs, pass down the bile-ducts and not rarely accumulate in the gall-bladder. They then pass into the intestine, where they promptly die and are digested. Only a few are passed into the outer world in a healthy condition (Schaper). The eggs are passed into the intestine with the bile, and are eventually expelled from the host.

According to Gerlach, the period spent by the distomes in the liver

Fig. 72.

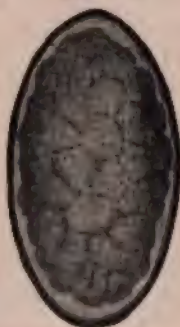


Fig. 71.

Fig. 71. Egg of *Distomum lanceolatum*.Fig. 72. Egg of *Distomum hepaticum*.

is from nine to twelve months. On the other hand, Leuckart and Friedberger hold that the parasites commence to migrate within three to four weeks after infection or immediately after they have become sexually mature. Schaper expressed the view that, like infection, migration of the parasites may take place during any part of the year.

The *Limnaeus minutus* or *truncatulus* is a small snail measuring about  $\frac{1}{2}$  cm. in length and provided with a brown, spiral shell. It occurs especially on marshy ground, in ditches and also in slow-flowing brooks. In the autumn it enters the water, preferably in a ditch with a clay bottom, and passes the winter there.

The life-cycle of the *D. lanceolatum* is in all probability similar but it is not yet known definitely. According to Piana, the pear-shaped larvæ penetrate the snail known as *Helix carthusiana*, and there develop into cercariæ. Leuckart believes that the escape of the embryos into the outer world is not spontaneous, but occurs in the intestine of a special intermediate host.

**Resistance of the Parasite.** The eggs remain capable of germinating from one year to another but do not resist drying or putrefaction. The larvæ, according to Friedberger, are not killed by night frosts, and the encapsuled cercariæ retain their power of development for weeks or even months (Leuckart). Salt solutions are very poisonous to the larvæ and cercariæ (Ercolani and Perroncito). A 2% solution is fatal in 5 minutes and a 1% solution in 20 to 35 minutes.

**Natural Infection.** As a rule infection results from the ingestion of green food carrying encysted cercariæ. This is especially likely to happen on moist and marshy land, and on land that is frequently flooded. Contaminated water must also be responsible in many cases. According to Lutz, the latter method is the common one in the Sandwich Islands. The cercariæ becoming detached from water-plants in shallow streams collect at places where the water is shallow, and are then taken up by the cattle when drinking. As some cercariæ tend to become encysted in the bodies of grass snails, infection may take place through the ingestion at pasture of plants bearing them. Spinola succeeded in infecting healthy sheep by feeding them with grass snails.

Housed animals may be infected, infection generally taking place through cercaria-infected green food, and sometimes through food from marshy land which has not been stored very long.

The disease is introduced into districts previously free, either by diseased domestic animals or by wild ruminants and hares.

Infection generally takes place in the summer or autumn, conditions then being favorable. According to Friedberger and Schaper, the infection may occur at any time of the year but, for reasons easily understood, infection is less severe in winter. Infection may occur during winter through the ingestion of food cut fairly fresh, or in "open" weather when the animals can go to pasture.

The degree of infection depends in the first place upon the extent to which the pasture, food and water are contaminated.

In this connection the dampness of the ground is of chief importance. The most severe infection occurs in places where the climate is damp or the rainfall copious, and the temperature comparatively high. Infection is also severe on flood land and during wet seasons. The period during which dangerous land is used as pasture, must also be taken into consideration. The longer the land is used the greater the number of flukes ingested. A stay of a few hours only, or even less than an hour, has in some instances led to serious outbreaks of the disease. In most cases repeated infection of the same animals is observed.

**Susceptibility.** Sheep and wild ruminants are the most susceptible, the ox is a little less so and the goat a little less than the ox. Swine, solipeds, and carnivora on the other hand rarely become diseased in spite of being invaded by the parasite. Young adult animals are more susceptible or their powers of resistance are less.

The wide distribution of the disease among sheep convinces Railliet that the cercariæ are to be found principally on the lower blades of grass which the sheep eat, whereas cattle get only the upper ones. According to Humble and Lush sheep with short lower jaws (parrot-mouthed) escape infection because they can only bite the tips of the leaves.

**Pathogenesis.** All the available evidence shows that the parasites, in migrating from the duodenum into the bile ducts, move by elongating their anterior end and fixing the oral and ventral sucker alternately. They spread rapidly through the liver until they reach the smallest bile ducts, where they are either arrested or may pass back again.

According to Gerlach, May and Spinola, the parasites gain access to the liver by penetration of the capsule after having passed out of the intestine through the wall. Luchs believes that the invasion of the liver is by way of the portal vein.

The flukes that have penetrated into the smallest ducts, burst through these at numbers of places, and passing into the liver tissue cause destruction of large areas of it and may consequently be frequently found under the serous membrane, or they may, by perforation of this, pass into the peritoneal cavity. As a result of the mechanical irritation, due particularly to the spiny covering of the parasites, and to the destruction of the liver tissue, an acute inflammatory condition is set up in both the bile ducts and the liver tissue. There may also be hemorrhages. Under certain conditions abscesses are produced in various parts of the liver. Schaper found streptococci and staphylococci in such abscesses. Flukes may pass into the portal or hepatic veins and in the latter case they may be carried to the right side of the heart and thence to the lungs or even to peripheral parts of the body.

The majority of distomes that have penetrated into the



liver tissue probably die or they may make their way back into the bile ducts, where they may become mature with those that have remained there. A chronic inflammation associated with the production of fibrous tissue is set up in the walls of the bile ducts. At a later stage this inflammation spreads to the interlobular connective tissue and results in cirrhosis of the liver. The destruction of liver tissue following the migrations of parasites also plays some part in the production of cirrhosis. The toxic products of the metabolism of the parasites not only cause irritation of the walls of the bile ducts, but also act upon the liver tissue after absorption into the lymph stream. Bacteria must also be taken into consideration in the production of cholangitis. These may either be carried out of the intestine by the flukes or they may be circulating in the blood and, reaching the liver, find conditions suitable for multiplication in the diseased walls of the bile ducts.

Jäger's investigations have shown that the absorption of the toxic material produced by the fluke is followed by a cellular infiltration in the angles between adjacent lobules. This in its turn is followed by a production of connective tissue and an extension of the process along the interlobular septa. As shown by Schaper an active proliferation takes place in the bile ducts by a process of budding.

The diffuse lesions of the liver tissue, the toxic metabolism products of the flukes and the bacterial infection cause disturbances of nutrition and of the process of blood formation. The susceptibility of animals affected with liver rot to specific infectious diseases may be increased (see hemorrhagic septicemia in Vol. I).

Carré and Bigoteau quite wrongly affirm that the symptoms of hydremia in cases of distomatosis are due to an intoxication brought about by the multiplication in some part of the body of the bacillus of pseudo-tuberculosis of Preisz and Guinard.

**Anatomical Changes.** If the invasion is extensive the liver shows evidence of an acute inflammation (Hepatitis acuta traumatica distomatosa). The liver is enlarged, hyperemic, its serous membrane is studded with small hemorrhages, and may be covered with a thin fibrinous deposit. The capsule shows here and there small, round, sharply-defined openings through which a dirty red liquid exudes on pressure, and sometimes the anterior extremity of a fluke may appear. These openings lead into irregular cavities in the liver tissue which contain blood, liver débris and numbers of young flukes embedded in a pulpy mass. If the liver be cut into, similar cavities are found in other parts. The large bile ducts are dilated and contain large quantities of muco-sanguinous bile and flukes in varying numbers. Exceptionally parasites escape into the peritoneum and set up a severe peritonitis, or there may be severe hemorrhage from the punctures caused by their escape.

In less severe cases of chronic distomatosis the liver may

appear healthy from the outside, but if palpated, hard cords can be felt. On section these are found to be dilated bile ducts with thickened and rigid walls. Pressure causes dirty, yellowish-brown bile to exude, containing flukes in variable numbers. Similarly altered ducts can sometimes be seen through the capsule, especially on the posterior surface, as white firm nodulated cords.

In more severe cases the liver becomes enlarged as a result of the chronic interstitial inflammation which appears later, and its consistency is at the same time firmer. The large bile ducts appear greatly dilated, their walls thickened and hard, and their inner surface is usually roughened by a deposition of salts (calcium phosphate and a little magnesium phosphate). In the lumen of the ducts greenish-brown, viscous bile and large numbers of flukes in various stages of development are found. In a single liver there may be 1,000 flukes or more. At a later stage there is shrinking of the liver, the organ becomes tough and the bile ducts appear as dilated tubes with calcified and thickened walls. Marked glandular proliferation of the mucous membrane of the bile ducts is almost always present (Schaper). As a result of repeated invasions various stages of the disease are often discoverable in a liver.

In severe cases there is usually ascites as well as general wasting and anemia.

If *D. lanceolatum* be present in very large numbers (they may number many thousands [Friedberger]), migration of the parasites may be followed by exactly similar symptoms, but as a rule the process is limited to the bile-ducts and the chronic lesions produced are not so severe as those described above.

According to v. Ratz the calcified nodules which sometimes occur in the livers of horses are due to flukes. In these nodules Ölt found the remains of young echinococci and Mazzantini the embryos of filaria. Morot found distomes in tubercle-like nodules in the peritoneum, and Cocu in blood-clots on the valves of the right side of the heart.

**Symptoms.** Unless the parasites are very numerous there are no functional disturbances, and a moderate invasion may cause no loss of condition. This has been shown over and over again in slaughter houses.

In acute cases, the period elapsing between infection and the appearance of symptoms is seventeen to twenty-one days. In chronic cases one to two months may elapse (Gerlach, Zürn, and others).

In the majority of cases, **sheep** show no symptoms for a month or two after infection, and the first symptoms seen are slight fever, dulness, weakness and anorexia. Careful examination will show that the hepatic region is painful on pressure and that the area of dulness is increased. The posterior border may extend beyond the costal arch and can be palpated (Spinola). Anemia due to a reduction in the hemoglobin content

and in the number of blood corpuscles may be demonstrated at an early stage and even in slight cases (Schaper).

In chronic cases, usually about the end of the autumn, there is edema. Simultaneously with the gradual paling of the mucous membranes there is edematous swelling of the conjunctiva which forms a prominent ring around the cornea, and causes swelling of the eyelid. Doughy swellings make their appearance along the trachea, under the brisket, and on the abdominal wall. By this time the dulness and weakness are pronounced, the appetite is suppressed, rumination slow, the wool dry and brittle and easily pulled out, and it even comes out spontaneously at places thus making the fleece ragged. Large numbers of fluke eggs can be found with the microscope in the feces. By this time there is evidence of hydrothorax and ascites.

About the third month of infection, generally at the beginning of winter, the wasting becomes more and more pronounced and the edema and ascites are more evident. The edema of the tracheal region decreases during the night and reappears when the animals are at pasture during the day, while the edema of other parts tends to decrease a little during movement and to reappear with rest. In addition to edematous swelling, Mergel observed abscesses on the neck and below the flank which he believed were due to flukes.

Coincident with the increasing emaciation, symptoms of anemia and cachexia become more and more prominent. In some cases there is diarrhea, the feces containing large numbers of eggs. The milk becomes watery and less nourishing, so that the lambs are poorly developed and may even die. Sometimes there is fever. Finally, the animals die from complete exhaustion. During the whole period of illness there is no jaundice or, at the most, very slight evidence of it.

More robust and particularly older animals recover gradually, especially after a moderate invasion, about the end of the winter or in the early part of the spring. The edematous swellings disappear, the appetite returns, and finally there is apparent complete recovery. The tissue lesions in the liver cannot be repaired and must eventually exercise a prejudicial effect upon the nourishment of the animal.

In **goats** the symptoms are very similar to those seen in sheep and diseased animals often abort, or their progeny are very weakly.

**Cattle, buffaloes and camels** present similar symptoms, but on account of their greater power of resistance the disease is as a rule, less severe. In these animals, edematous swellings along the course of the trachea and in the dewlap are more rarely seen, but there may be digestive disorders such as anorexia, diarrhea, and tympanites and, in severe cases, anemia and emaciation. A larger proportion of animals recover, and



the disease may remain latent, although the infestation may be severe (Wiegel). In exceptional cases, and especially among young animals, death is due to utter exhaustion. According to Vaelzen and Pease, many buffaloes are slaughtered in the East Indies on account of distomatosis.

Very occasionally, the disease runs the same course in **solipeds** (ass) as in ruminants, wasting, anemia, dropsy, and in some cases jaundice being observed (Prietsch).

In **rabbits** there is rapid wasting, hydrothorax, and ascites and, in occasional cases, swelling and jaundice of the mucous membranes (Braun).

**Course.** Acute cases in which sheep die in seven to nine days after the onset of symptoms from acute hepatitis, are only rarely encountered (Bonvicini). In some cases, death is quite sudden, possibly owing to embolism of young flukes in the vessels of the brain (Gerlach) or to hemorrhage into the peritoneum following perforation of the capsule of the liver. With such exceptions the disease tends to become chronic. The course of the disease is divided by Gerlach and Zündel into four stages. (1) The stage of traumatic hepatitis which begins soon after infection, lasts four to thirteen weeks, and is characterized by slight symptoms of fever, digestive disorders, and enlargement and sensitiveness of the liver. (2) The stage of chlorosis. This sets in in the autumn, lasts six to twelve weeks, and is characterized by symptoms of anemia. (3) The stage of wasting as a rule sets in about four months after infection during the winter months. In this stage emaciation and dropsy are pronounced and deaths are frequent. (4) The migration of the flukes occurs in the spring, about May or June (Gerlach). During this stage, symptoms disappear and there may be temporary or permanent improvement. The investigations of Friedberger and Schaper have shown that this division into stages is applicable to a certain extent in cases where there are extensive outbreaks because there are a number of animals severely and simultaneously affected. Such outbreaks generally occur in the summer and autumn.

In most cases the stages cannot be marked off from each other and the series of symptoms described above cannot be followed out. The eggs of the parasites may be passed out and the parasites themselves migrate at any time. There is great variation in the duration of the disease. In some cases where the infestation is severe, it may last only three months, and in another case it may extend to a year or more.

**Diagnosis.** Obvious symptoms are seen in advanced cases only, and more or less closely resemble those due to other causes (tapeworms, gastric strongyles, etc.). The edematous infiltration cannot be considered as characteristic of liver rot. As a rule, no difficulty is experienced in practice, in arriving

at a diagnosis when there is a dead animal available for examination. During life a certain diagnosis can be based upon the discovery of the eggs of flukes in the feces. It must be remembered that in the early stages of the disease there may be no eggs in the feces and in such cases, enlargement and sensitiveness of the liver should arouse suspicion as to the existence of liver rot. This suspicion may be supported by a history indicating the likelihood of infection.

Microscopic examination of the feces can be carried out by diluting and sedimenting the feces, or better still by repeated washing and filtering through a gauze filter until the yellow color disappears (Lutz). The number of eggs in the feces is very variable, depending probably upon the quantity of bile passing out of the liver. According to Perroneito one finds an average of 10 eggs in each preparation if 800 flukes are present, and Brusaferro puts the figure at 1 to 13 if 100 flukes are present. In severe cases, however, there may be as many as 30. The feces of six young bovine animals badly affected with distomatosis were systematically examined by Hutyra & Marek, adopting Lutz's method, and only isolated eggs could be discovered.

The eggs of the *D. lanceolatum* are found far more rarely, so that examinations may be negative although there may be large numbers of the parasites in the liver.

**Prognosis.** If the symptoms are pronounced, prognosis is unfavorable. The symptoms may abate from time to time, but there is scarcely ever a complete recovery. Young and weakly animals are generally affected more seriously than older and more robust ones, and cattle survive infection better than sheep.

**Treatment.** No drug has as yet been discovered that is of any use. All that can be done is to nourish the animals as well as possible (good hay, corn, bran, oil cake, etc.). As a rule, well-nourished animals offer more resistance than poor, debilitated ones. If the animals are anemic or show symptoms of edema, they should be slaughtered as soon as possible.

The following drugs have been tried: benzine in doses of 5 to 6 gm. or for cattle 60 to 120 gm. for 4 to 6 days (Bunck); naphthalin twice daily for a week in doses of 0.7 to 0.1 gm. (Mojkovszki); turpentine (Perroneito); tincture of iodine (de Romanet); creosote, pierate of potash, carbon bisulphide (Flóris).

**Prophylaxis.** The simplest and best known prophylactic measure among farmers is the avoidance of moist, low-lying land, especially for young animals. If this be impracticable suitable land not being available, or on account of wet weather, sheep should be given seven to eight grammes of salt (daily) mixed with hay before they are turned out, and if possible, their drinking water should contain salt in the proportion of one-half per cent. Trasbot states that pine shoots mixed with bran are useful, and Cadéac advises leaves. Further it appears to be advisable to give the animals plenty of room so that they may not be forced to eat the lower parts of the grass (see page 549). It has been advised to sprinkle the fields with salt and lime(?).

In order to reduce the risk of infection it is advisable to improve the pastures by drainage. Care should be taken that the livers from diseased sheep and cattle be destroyed, or, if given to dogs they should be cooked since living eggs may be scattered over the land with the feces of such animals. Schaper advises that affected animals should be housed or, at least brought into enclosures on hard dry ground in order to lessen the possibility of fresh infection of the pastures. The manure of the animals can then be best used for arable land. Finally, water snails should be collected and destroyed.

**Literature.** Albanese, Rev. Gén., 1908, XI, 147 (Rev.).—Braun, Kaninchenkrkh., 1907, 67.—Cadéac, Rev. Vét., 1885, 10.—Carré & Bigoteau, Rev. Gén., 1908, XI, 433.—Friedberger, D. Z. f. Tm., 1878, IV, 145.—Gerlach, Ger. Tierheilk., 1872, 487.—Jäger, A. f. Tk., 1906, XXXII, 410.—Leuckart, Parasiten d. Menschen, 1879, 3-5 Lief., 1-5, 34.—Lutz, Cbl. f. Bakt., 1893, XIII, 320.—Pfeiler, Z. f. Fleischhyg., 1907, XVII, 174.—Prietsch, S. B., 1906, 64.—Schaper, D. Z. f. Tm., 1890, XVI, 1 (Lit.).—Thomas, The Vet., 1883, 180, 469.—v. Velzen, Vet. Jhb., 1890, 91.—Wedernikow, A. f. Vet.-Wiss., 1893, 143.—Wolffhügel, Z. f. Infkr., 1907, II, 546.—Zündel, La distomatose, 1880.—Zürn, Tier. Parasiten, 1882, 207.

**Other Distomes.** In addition to the two types described, the following distomes occasionally occur in the domestic animals. *Distomum truncatum* Rud. (*D. conus*, *Amphistomum conicum*), which according to Braun is identical with the *D. campanulatum* of Ercolani, and *D. felineum* Riv. Both of these occur now and then in the livers of cats and dogs (Creplin, v. Ratz, de Jong, Zwaardemaker, Rivolta, Ercolani) and if present in large numbers cause dilatation of the bile-ducts and chronic hepatitis. *Distomum conjunctum* was found by Lewis enclosed in cysts or purulent centers in the livers of dogs in India. According to Braun, the *D. albidum* occurs only in cats. *D. magnum* is found in cattle in Texas (Stiles & Hassal). *Monostomum hepaticum* has been found in pigs.

**Literature.** Braun, Cbl. f. Bakt., 1893, XIV, 465.—v. Ratz, Z. f. Flhyg., 1900, X, 141.—Stiles & Hassall, The Insp. of Meats, 1898.—Zwaardemaker, V. A., 1890, CXX, 197.

### (c) Cysticercosis of the Liver.

#### (*Hepatitis cysticercosa.*)

Cysticercosis of the liver is an acute or chronic hepatitis caused by the *Cysticercus tenuicollis* or *C. pisiformis* with secondary peritonitis.

**Occurrence.** Cysticercosis of the liver is seen principally in lambs, young pigs and rabbits or hares. It is also seen in calves (Falk) and in the cow (Pütz). In the first mentioned species it usually occurs as a local disease and causes heavy losses. Almost without exception animals between the ages of a few weeks to several months are affected.



Enzootics of cysticercosis of the liver have been observed by Kühnau, Brusafarro, Avéradère and Moussu. Similar outbreaks have been described in young pigs by Dürbeck and Seiler. The latter authors have made a special study of the pathological anatomy of the disease.

**Etiology.** In cattle and pigs the disease is caused by the *Cysticercus tenuicollis*, which is the cystic stage of the *Tænia marginata* which inhabits the intestine of the dog (see page 468). In rabbits and hares the cause is the *Cysticercus pisi-formis*, the cystic stage of the *Tænia serrata* also an inhabitant of the intestine of the dog (see page 468).

Infection occurs naturally through the ingestion of feces of dogs harboring these parasites. The proglottides or the contained eggs are rarely dropped directly into feeding- or drinking-troughs. As a rule, the feces are dropped about on the ground or in the neighborhood of drinking places and thus contaminate the food or water. Pigs may ingest them while rooting.

**Susceptibility.** It cannot be determined whether there is any difference of susceptibility among animals that harbor the *Cysticercus tenuicollis*. The varying incidence of the disease in the different species may be due to differences of surroundings. Age has some influence upon the susceptibility, since young animals are attacked more frequently and more severely. The fact that animals are affected at the age of a few weeks, shows that within that time the opportunity offers of ingesting the eggs of tapeworms. Young pigs may also ingest the eggs while rooting.

**Pathogenesis.** The embryos of the *Tænia marginata* and *T. serrata* are liberated from the eggs by the acid stomach contents, and reaching the intestine bore through the wall and enter the portal veins by which they are carried to the branches of the portal vein in the liver. They have been found there four hours after the ingestion of the eggs (Hoffmann). Some embryos may pass through the capillaries into the hepatic veins, and thus be carried to the lungs or other organs. After their arrest in the blood vessels they bore their way out, and penetrating the liver tissue, make their way to the surface leaving tracks behind them. Finally, some of them perforate the capsule of the liver and reaching the peritoneal cavity develop further in the omentum, mesentery, or peritoneum. Those that remain under the capsule of the liver also undergo processes of development. The same thing happens to the embryo arrested in the vessels of the lungs.

A severe invasion causes a condition similar to that produced by a heavy infestation with flukes (see page 549). There is an acute hepatitis often associated with hemorrhage and a localized or wide-spread peritonitis. The rupture of large vessels may cause fatal hemorrhage. If the animals survive, the acute hepatitis is followed by an interstitial inflammation,

causing cirrhosis. In some cases the embryos in the lungs set up broncho-pneumonia and even pleurisy.

**Anatomical Changes.** In the acute stage the liver is more or less enlarged, although there may be no enlargement in some cases. Its serous membrane is dull and may be covered with a hoar-like fibrinous layer. In many cases the surface of the organ appears nodular and the apex of each nodule shows a minute opening. The liver itself has a speckled, mosaic-like appearance (mosaic liver or trout liver) on account of the variation of color of the lobules. Some of these are black or blackish-red and others are pale red or grayish-brown. The darkly colored lobules are enlarged. The consistency of the organ is soft, and the tissue is easily broken down. Close examination sometimes shows within the dark-colored lobules yellow points or transparent acephalic cysts measuring three to four mm. in length, by one to two mm. broad. In the later stages there can be seen burrows in the liver tissue which at first were filled with blood and afterwards become yellowish-gray in color as a result of degeneration of the injured tissue, and the outpouring of an exudate. These burrows are sometimes sinous and sometimes wider at one end.

Microscopic examination reveals the presence of cysticerci in the little pools of blood and cellular infiltration in the neighborhood and in the interlobular connective tissue, especially at the angles of the lobules. Finally there is compression and partial degeneration of the liver tissue.

Not rarely there is acute peritonitis or there may be liquid blood in the peritoneum. In this blood may be seen numbers of very minute pale-colored bodies (young cysticerci).

In chronic cases there is more or less chronic interstitial hepatitis and shrinkage of the new connective tissue.

**Symptoms.** The symptoms of acute cysticercosis of the liver set in a few days after infection. This has been proved by the feeding experiments of Küchenmeister, Leisering, Leuckart, Baillet and Railliet. A kid infected experimentally by Railliet became seriously ill nine days later. The severity of the infection influences the duration of the period of incubation, symptoms setting in later when the infection is not very severe.

In young pigs the course of the disease is sometimes very acute, animals dropping dead without apparent cause. In other cases the symptoms are depression, staggering gait, and marked dulness. Death may be sudden.

As a rule the evolution of the disease is more gradual. Dulness, great weakness, inappetence, and thirst may be observed, and in the later stages acute peritonitis with fever, distension of the abdomen and sensitiveness of the abdominal wall. In such cases there is rapid emaciation. Death may occur with-

in a few days or the animals may live for some weeks until there is marked anemia and debility.

**Chronic Cysticercosis.** This is the form of the disease usually seen in the rabbit, but it occurs also in other species of animals. The symptoms are chronic digestive disorders which cause some loss of condition.

**Diagnosis.** The specific nature of the disease is best shown by the demonstration of young cysticerci in liquid withdrawn from the peritoneum. As a rule a diagnosis is only possible post mortem, although a strong suspicion as to the nature of the disease may be raised if there are a number of young animals showing the symptoms described, and especially if the surrounding circumstances are favorable to infection. In very recent cases or when there are no burrows in the liver, tissue diagnosis may be attended with difficulty even at the autopsy, especially if no microscopic examinations are made. In such cases other forms of acute hepatitis and other infectious diseases (swine erysipelas, hemorrhagic septicemia) have to be excluded. Acute distomatosis is more easy to diagnose because of the presence of young distomes which measure at least one to three mm., either in the burrows in the liver tissue or in the peritoneum.

**Prognosis.** Prognosis depends entirely upon the severity of the infestation, but in acute cases it is unfavorable. Avéradère records a mortality of 38 per cent in an outbreak among lambs. Chronic cases are fatal far more rarely, especially in adult animals, and as a rule there are no symptoms in these cases.

**Treatment and Prophylaxis.** Treatment must depend upon the nature of the case. As a prophylactic measure all dogs in the neighborhood of the animals and especially sheep dogs, should undergo a course of treatment for tapeworms two or three times a year or, if possible, should be kept quite away from the animals.

**Literature.** Avéradère, *Rev. Vét.*, 1898, 333.—Brusaferro, *Clin. Vet.*, 1893, 214.—Dürbeck, *Monh.*, 1899, X, 32 (Lit.).—Engel, *W. f. Tk.*, 1878, 165.—Falk, *Z. f. Flhyg.*, 1898, VIII, 93.—Hoefnagel & Reeser, *D. t. W.*, 1905, 444 (Rev.).—Hofmann, *B. t. W.*, 1901, 537.—Kleinpaul, *B. t. W.*, 1907, 131.—Moussu, *Rec.*, 1902, 657.—Neumann, *Mal. parasitaires*, 1892, 484.—Pütz, *Z. f. pr. Vet.-Wiss.*, 1876, 169.—Railliet, *Zool. méd.*, 1895, 229.—Seiler, *A. f. Tk.*, 1903, XXX, 339 (Lit.); *D. t. W.*, 1907, 436.

**Other Animal Parasites Found in the Liver.** The following parasites are sometimes clinically important as occurring in the liver:

1. The larvæ of *sclerostomes*. These reach the liver by way of the portal blood and make burrows in the liver tissue in a similar manner to cysticerci (Colucci, Mégnin, Schlegel). Schlegel repeatedly saw cases of chronic hepatitis which clinically resembled Schweinsberg disease, (see page 530), in which the liver was enlarged two to three



times, and showed small nodules varying in size up to a pin's head, especially close under the capsule. In these cases there were also narrow tortuous burrows in the liver tissue of a red or reddish yellow color. The contents of the nodules and burrows were either caseous or calcified, but the larvæ of sclerostomes could be demonstrated.

2. **Eustrongylus gigas.** This parasite was found by Lissizin in the liver of a dog which had had convulsions for three days.

3. **Linguatula denticulata** (*Pentastomum denticulatum*) was found in large numbers in the liver of a goat that had shown symptoms of debility for a long time. The same parasite was found in the liver of an otherwise apparently healthy ox. The capsule of the liver showed large numbers of openings measuring about 3 mm., and leading into cavities each of which contained a larval linguatula. Similar cavities were visible in the deeper layers of the liver.

4. Occasionally **ascarides** (*Ascaris megalocephala*, *A. suilla*) wander out of the intestine into the large bile-ducts in the horse and pig and may cause symptoms of colic or epileptiform seizures (Röll, Ortmann). In cats the embryos of the *Ollulanus tricuspis* which normally inhabits the stomach are sometimes found in the liver.

**Literature.** Gerlach, Hann. Jhb., 1869.—Lungwitz, Z. f. Flhyg., 1893, III, 218.—v. Rätz, Vet., 1890, 269; 1892, 305 (Lit.).—Schlegel, B. t. W., 1907, 53.

#### (d) Coccidiosis of the Liver. *Coccidiosis hepatis.*

Coccidiosis of the liver is an enzootic disease among rabbits, affecting the liver and the bile ducts and is caused by the *Coccidium oviforme*.

**Etiology.** The *Coccidium oviforme* Leuckart (*Eimeria cuniculi*) has a double outer membrane with one pole slightly flattened and measuring 30 to 50  $\mu$  in length, and 14 to 28  $\mu$  in breadth. (For the development of coccidia, see page 497.)

**Natural Infection.** The natural method of infection is by the ingestion of food or water contaminated with the feces of rabbits that are affected with the disease or that have coccidia in their livers. Young animals are especially susceptible, and in them the disease is apt to be severe. Adult animals, on the other hand, are affected less seriously, and as a rule, show no symptoms. Such animals are capable of infecting young animals by means of their feces.

**Pathogenesis.** It is not yet known in what form and in what manner the parasites reach the liver. They multiply in the bile ducts and produce enormous proliferation of the epithelium.

**Anatomical Changes.** The lesions vary in size up to a pea or even a hazelnut, and take the form of whitish-yellow nodules that are somewhat prominent and contain a creamy or cas-

eous material. There is a connective tissue capsule. Coccidia are found in enormous numbers in the content of the nodules.

**Symptoms.** The symptoms are disturbance of nutrition, emaciation, weakness and, later, staggering gait, inappetence and eventually jaundice of the mucous membranes. In the last stages one observes diarrhea which is very persistent, a discharge from the nose, and finally convulsions. Young animals usually die in about two to three months, and whole warrens are often wiped out.

**Diagnosis.** It is quite easy to arrive at a diagnosis during life by the demonstration of coccidia in the feces by means of the microscope. The feces contain fewer coccidia than in cases of intestinal coccidiosis. The two conditions are often observed in the same animal.

**Treatment and Prophylaxis.** Treatment is of no avail and efforts must be directed principally towards prevention. In this connection attention must be paid to the separation of the young animals from the adults as soon as possible, all visibly diseased animals must be destroyed, hutches must be disinfected, kept dry and well ventilated.

Coccidia have been found by Johne in cavities as large as apples in the liver of a pig. Perroncito and Rivolta record their occurrence in the livers of dogs, and they were found by Chierici in a cat's liver that was somewhat enlarged and showed signs of chronic cholangitis.

**Literature.** Johne, S. B., 1881, 60.—Neumann, *Mal. parasites*, 1892, 488 (Lit.).—Railliet, *Zool. méd.*, 1895, 134.—Zürn, *Vortr. f. Trztle.*, 1878, I, H, 2 (Lit.).

**Protozoal Hepatitis of the Pigeon** (Caseous hepatitis of the pigeon). Young pigeons sometimes die suddenly as a result of multiple caseous lesions in the liver without having shown any symptoms. In such cases the liver is enlarged and contains large numbers of grayish-yellow dry caseous foci. The foci vary in size from a millet seed to a hazelnut and are irregularly rounded in shape. Sero-fibrinous peritonitis is also present. The disease was first described by Rivolta in 1878 and later by Jowett (1907). Both authors found protozoa in the liver lesions provided with one or two flagella. This parasite was named the *Cercomonas hepatis* by Rivolta. In 1910 two cases were studied carefully by v. Ratz who found that the parasites were provided with three flagella and came to the conclusion that the parasite was the *Trichomonas columbæ* which usually inhabits the intestine or respiratory organs of the pigeon. Under some circumstances which are not known they make their way out of the intestine and penetrate the liver, there setting up nodular necrotic lesions.

He believes that the parasite found by Rivolta and Jowett was the *Trichomonas* (v. Rätz, Közl., 1910, VIII, 184).

## SECTION VII.

### DISEASES OF THE PANCREAS.

References to diseases of the pancreas in the domestic animals are very scanty in literature. This is in part due to the fact that clinical investigation of the gland is impossible in the case of herbivora and possible to a slight extent only in the carnivora. The special methods of investigation that have been employed to obtain exact information regarding the digestive phenomena have not been undertaken from a veterinary point of view. It is chiefly in the horse, dog and cat that diseases of the pancreas have been observed.

**Symptoms.** The fact that there may be no digestive symptoms in a case where there is a disturbance of function of the pancreas is due to the circumstance that to a certain extent the pancreatic juice can be replaced by the other digestive juices and by the intestinal bacteria. Disease of the pancreas may show itself in two ways. In the first place, the hydrolysis and absorption of fat is incomplete and in consequence the feces contain a comparatively large proportion of fat (stearrhea). In carnivora the feces are gray in color and have a characteristic greasy appearance. According to Müller, the proportion of free fatty acids and soap present is considerably reduced on account of the imperfect decomposition of the fat. In the second place, sugar metabolism is deranged, with the resulting production of diabetes mellitus both in experimental animals and in natural cases of disease of the pancreas (see Vol. I).

In certain diseases the pancreas can be palpated through the relaxed abdominal wall and in such cases valuable information may be gained, especially in carnivora. Nevertheless it is extremely difficult to differentiate between pancreatic lesions and tumors of the pylorus or enlarged portal lymphatic glands. This difficulty is also present in cases where the enlargement of the gland has caused jaundice or ascites by pressure on the bile ducts or portal vein.

Mégnin & Nocard described a **catarrh of the pancreatic duct**. The duct was completely occluded by a catarrhal secretion with the result that there was a production of connective



tissue, subsequent shrinkage of which led to atrophy of the gland parenchyma. Catarrh of the bile ducts and chronic interstitial hepatitis were present at the same time. During life the symptoms shown by the horse were: jaundice, partial loss of appetite, languor, unsteady gait and emaciation. The feces were dry and pale in color. In two months the animal was in a state of complete collapse.

**Chronic Inflammation of the Pancreas.** This was observed by Siedamgrotzky in a horse, the animal showing loss of hair, edema, leucocythemia and debility. The stroma of the pancreas was greatly increased in amount, and the gland tissue reduced, and the ducts were obstructed with viscid mucus and calcified flakes. (Kitt found a similar lesion in a horse.) In a case recorded by Wheatley, there was extensive cirrhosis of the gland which weighed sixteen pounds, the ducts were dilated and filled with material resembling albumen. The horse had shown gradual wasting, edema and excoriation of the skin at places.

**Suppuration of the pancreas** may be set up by the presence of foreign bodies in Wirsung's canal. In a case recorded by Goubaux in the horse, the foreign body was a straw stalk, and in the dog, Bruckmüller records the presence of a needle in the canal. The symptoms presented were loss of appetite, emaciation, occasional attacks of colic and susceptibility to pressure in the hypogastric region. Schättler found hemorrhages and a number of small abscesses in the pancreas of a horse, caused by the penetration of a piece of wire from the duodenum. The symptoms shown were: loss of energy, frequent groaning, and variable appetite.

**Nodular fat-necrosis of the pancreas** occurs in absolutely normal lobules in fattened pigs (Marek, Rónai). It also occurs exceptionally in the dog and horse. No symptoms are produced and in cases where they cause sudden death they may be associated with multiple hemorrhages of the gland. Hemorrhages in the pancreas and surrounding tissues were found in two cases by Prettnner and in one case by Mettam, in dogs which had shown symptoms of vomiting, abdominal pain and sudden collapse. Prettnner is inclined to think that the condition was due to an infection from the intestine. In Mettam's case the gland was enlarged to a considerable extent and beset with necrotic foci. In one case similar lesions were found in a horse that had died from acute enteritis of the small intestine and peritonitis.

**Atrophy of the pancreas** was found by Liénaux, Eber and Sendrail in dogs dead of diabetes mellitus. In Liénaux's case the pancreas was reduced to 3 cm. in length and 1 cm. in width,

and in Sendrail's case it was shrunken to the size of a bean. In a case of atrophy of the pancreas in a dog observed by Müller there was emaciation in spite of the appetite being maintained; undigested muscle fibers were found in the feces and there was an absence of free hydrochloric acid in the stomach.

Mention must also be made of the following abnormalities that have been met with at postmortems. Calculi in the ducts of the gland cause dilatation of the ducts, an increase of connective tissue, and atrophy of the parenchyma. Calculi of the pancreas have been met with fairly frequently by Scheunert & Bergholz, especially in the cow. The presence of the following neoplasms has been recorded: adenoma (Liénaux), carcinoma of the head of the pancreas, especially in the dog, and melanoma in the horse (Bruckmüller, Friedberger, Käsewurm). Echinococci, larval sclerostomes and intestinal worms have occasionally been observed. Nencioni found chronic pancreatitis in an emaciated cat due to a colony of *distoma felineum* in the gland.

**Literature.** Goubeaux, Rec., 1875, 807.—Guérin, Bull., 1906, III.—Marek, D. Z. f. Tm., 1896, XXII, 408.—Mégnin & Nocard, Arch. d'Alf., 1878, 601.—Mettam, The Vet., 1901, 619.—Müller, Dresd. Ber., 1906, 162.—Nencioni, N. Erc., 1906, 26.—Prettner, T. Z., 1894, 342.—Reimers, Ann., 1887, 672.—Rónai, Hússzemle, 1906, 33 (Lit.).—Schättler, D. t. W., 1905, 206.—Sendrail, Rev. Vét., 1906, 229.—Scheunert & Bergholz, Z. f. physik. Chemie, 1907, LII, 338.—Siedamgrotzky, S. B., 1878, 30.—Wheatley, Journ. of Comp. Path., 1896, 44.

## SECTION VIII.

### DISEASES OF THE PERITONEUM.

#### 1. Ascites. Hydrops ascites.

Ascites is the term applied to the collection of serum-like fluid in the peritoneum not due to an inflammatory process.

**Occurrence.** Ascites occurs most commonly in the dog and generally as a result of disease of the heart or liver. The disease is seen in ruminants far more rarely, with the exception of the general dropsy seen in cattle fed on products of sugar factories. It is only quite exceptionally that the disease is observed in the horse, pig and birds, and then it is generally a condition accompanying either chronic disease of the liver or the peritoneum.

The disease was found in  $\frac{1}{2}\%$  of 70,000 dogs examined by Fröhner. Cadiot saw 37 cases in three years at Alfort. Out of 28 cases 10 were due to disease of the heart and pericardium, 8 to tuberculosis, 4 to pleurisy, 2 to malignant growths in the liver and lungs, 3 to chronic interstitial hepatitis and one to cancer of the liver.

**Etiology.** The principal cause of ascites unassociated with general dropsy of the rest of the body and of the subcutaneous tissue is congestion of the portal area. This congestion may be due to compression of the portal vein by neoplasms, enlarged lymphatic glands or tumors of the pylorus or of the head of the pancreas. Under these and other conditions there is thrombosis of the portal vein. Portal congestion may also be set up by chronic diseases of the liver such as cirrhosis, echinococcosis, and neoplasms. In these cases the interlobular branches of the portal vein are subjected to pressure and the outflow of blood from the portal area is thus obstructed.

In cattle ascites is due most commonly to tuberculosis. Other chronic diseases of the peritoneum, such as neoplasms, may have a similar effect. These growths may cause obstruction of the mesenteric or portal veins or of the great lymph vessels and prevent the absorption of the lymph. The more common sequel to these lesions is chronic peritonitis.

As a part of general dropsy ascites occurs in cases of thrombosis or compression of the posterior vena cava between the liver and the heart, heart disease and chronic diseases of



the lungs. Although in these cases ascites is the result of passive congestion of the venous system throughout the body, or at least in the hind quarters, it very often remains the only symptom for a long time, especially in dogs.

Ascites and other symptoms of dropsy are often seen in animals that are cachectic. A similar form of the disease is seen in cases of chronic nephritis, liver rot, parasitic pneumonia and gastritis, or in cases where substances rich in water figure largely in the diet (turnips).

In young animals, and especially in young dogs, uncomplicated cases of ascites apparently occur. The fact that such cases often recover completely, suggests the uncomplicated nature of the condition.

According to Hamburger this is a specific condition caused by an organism named by him the "Bacterium lymphagogen" and which increases the quantity of lymph through the action of its metabolic products.

**Anatomical Changes.** In the horse there may be as much as 170 liters of liquid in the peritoneum (Brusasco) and in the dog twenty liters (Hördt). The liquid is sometimes clear as water and in other cases slightly turbid. It may be faintly yellow in color, watery, and contains at most only traces of fibrin. The specific gravity is below 1,016, but, as a rule, very nearly that; it contains 3.5 per cent of albumin and its chemical reaction is either alkaline or neutral. The fluid is often opalescent. By transmitted light it may have a yellowish tint and by reflected light a greenish color. This is probably due to the presence of blood pigment. When the transudation is due to a rise of pressure in the portal system the liquid may be from reddish to blood red in color. In cases of jaundice it is greenish and bile pigments can be demonstrated in it. There is, as a rule, only a small amount of sediment composed of a few white blood corpuscles, desquamated endothelial cells infiltrated with fat, granular debris, threads of fibrin, and not rarely a few red blood corpuscles. In some cases the latter are present in large numbers (Fig. 73).

The peritoneum appears in some cases smooth and glistening, but where the disease has been in existence for a long time it is thickened and shows adhesions at places. The abdominal viscera appear anemic and even atrophied. In many cases the postmortem examination is completed by the discovery of some primary condition.

In cats and dogs the transudate is sometimes comparatively rich in fat (Ascites adiposus or chyliformis) and appears gray or milk white. The liquid may be translucent or quite opaque and when allowed to stand a thick layer of fat may form on the surface. The fat droplets are derived from desquamated endothelial cells that have undergone fatty degeneration or from the cells of neoplasms (cancer, epithelioma), or directly from the blood in cases of lipemia. Wohlmuth found white colored serum in the peritoneal cavity of a fowl.

In many cases the fluid resembles lymph (ascites chylosus). Cases of this kind have been observed in cats by Dollar, Gray, Marcone, Wohlmuth and Suffran, and one case in the dog by John. The lymph may be derived from a ruptured

thoracic duct or from the lymph vessels of the intestinal wall as a result of peritoneal carcinoma. Nothing special could be found in the peritoneum in Wohlmuth's case. The fluid is milk-white, it contains a large number of lymph corpuscles, and on standing a creamy layer forms composed of fat droplets. Storeh found that the liquid obtained from Wohlmuth's case was alkaline (in contradistinction to the amphoteric reaction of milk), the specific gravity 1015, the fat droplets were very minute and the percentage of fat was 7.6. There was a little sugar, fibrin, globulin and albumin.

Bennert, Joachim and others have shown that the milk-like appearance of transudates that occur in the peritoneal cavity of man is sometimes due to a pseudoglobulin which contains lecithin. In such cases the addition of ether and potassium hydrate does not cause the liquid to clear up.

**Symptoms.** The presence of large quantities of liquid in the peritoneum is evidenced by a distension of the abdomen. In emaciated animals this distension appears larger than it really is as it is in contrast with the wasted quarters. The more or less enlarged abdomen (Fig. 74) varies in shape according to as it is looked at from before or behind. In the downward di-



Fig. 73. Sediment of ascitic fluid. a. Endothelial cells infiltrated with fat, b. white corpuscles, c. red corpuscles.

rection it gradually increases and reaches its maximum in the neighborhood of the white line. The flanks fall in and the transverse processes of the lumbar vertebræ and the last ribs become prominent. In some cases the spines of the lumbar vertebræ form a convex line. In small short-coated animals the abdominal wall stands out behind the costal arch because this offers a greater resistance to the pressure of the liquid than the soft abdominal wall. The lower line of the abdomen becomes horizontal (Fig. 75) or curves downwards and extends below the line of the sternum (pendulous belly). In the dog the abdomen sometimes comes in contact with the ground and the umbilicus may appear as a hemispherical translucent projection. The

shape of the abdomen varies with the position of the animal. If a small animal be placed on its hind legs or in a sitting position the pelvic portion of the abdomen becomes prominent (Fig. 76). If the dorsal position be taken up, there is bulging of the flanks. Difficulty of respiration follows if the animal be held on its fore legs because the fluid presses on the diaphragm and hinders its movements.

On palpation the upper parts of the abdominal wall are found to be more or less tense, but the lower part is yielding. If the abdominal wall be not too tense intermittent pressure sets up undulating movements in the liquid which can be felt with the hand or even seen in some cases. In the larger animals the movements of the liquid can be appreciated by a hand introduced into the rectum.

Percussion reveals dulness extending upwards to various levels and the upper limit is sometimes marked by an undulating line. In small animals set up on their hind feet the dulness is found in front of the pelvis in the pubic region and if the animal be on its back, in the region of the loins or flanks.

In the larger animals auscultation over the lower part of the abdominal wall reveals no intestinal murmurs or very faint ones. The position of the area of dulness varies with the attitude taken up by the animal. By tapping the abdominal wall smartly with the flat of the hand or by shaking the animal's body, splashing sounds are sometimes produced which resemble those produced by the displacement of liquid gastric or intestinal contents containing large quantities of gas.

The extent to which the respiration is impeded depends upon the quantity of liquid. In order to take as much pressure as possible off the diaphragm and lungs, the animals, and especially dogs, prefer to take up a sitting position and to avoid all movement.

The compression of the stomach and intestines by the liquid causes constipation, tympanites and anorexia. In dogs the persistent pressure on the bladder sometimes causes the involuntary passage of urine (Spinola).



Fig. 74. Distension of the abdomen owing to ascites.



to feel the end of the cystlike body and, with very few exceptions, there is no change in the shape of the abdomen or in the position of the area of dullness in different positions of the animal. The loops of the intestines do not permit of any change of position of the large growth at all or only after some time. In the larger animals rectal examination furnishes valuable evidence for the exclusion of tumors or pregnancy. Retention of the urine is almost always observed in cases of distension of the bladder. Exploratory puncture is negative when there is a tumor. In cases of cystic ovary a viscid liquid containing ciliated epithelium is withdrawn and in hydro- or pyometra the liquid is generally turbid or puslike and putrid. The differential diagnosis of obesity is easy, because alteration of the position of the animal does not affect the shape of the abdomen and extensive areas of dullness are lacking. Of course, the negative result of exploratory puncture requires an explanation.

The presence of liquid in the peritoneum is not diagnostic of ascites, because the same condition is met with in serous peritonitis and as a result of rupture of the bladder. The differential diagnosis of acute peritonitis is easy on account of the pain on pressure on the abdominal wall, the elevation of temperature and the rapid course of the disease. It is more difficult to exclude the possibility of chronic peritonitis. The symptoms so closely resemble each other that a well-founded di-

agnosis can be based on the examination of the liquid obtained by puncture in cases where the primary disease and the manner of development of the condition are not known. Contrary to what is the case with the transudate (see page 565) the exudate is turbid, contains a large proportion of albumin (more than 3.5 per cent); its specific gravity is rather high (above 1.016) and it contains fibrin and is comparatively rich in white blood corpuscles. In cases of rupture of the bladder, the bladder is found to be empty and the expired air, the skin and the liquid obtained by puncture smell of urine.

It is often difficult to discover the immediate cause of the ascites. As a general rule simple ascites is due to some dis-



Fig. 76. The distension of the pelvic and neighboring portion of the abdomen due to ascites, the animal being held up in a sitting position.

ease in the portal area, while dropsy of the other body cavities and of the subcutaneous connective tissue indicates stagnation of blood in the vena cava due to disease of the heart, lungs or kidneys, or it may indicate general hydremia. Exceptions to this rule are not uncommon. The pressure of the transudate on the blood- and lymph-vessels of the abdominal wall may cause subcutaneous edema. Diseases of the heart and lungs not rarely cause symptoms which for a time resemble those of ascites. A careful examination of all the organs allows some opinion to be formed as to the situation of the primary disease. The endocardial murmurs, irregularity of the heart and pulse are sufficiently diagnostic of diseases of the heart. A decrease in the quantity of urine passed is easily observed. The differential diagnosis of chronic diseases of the lungs is fraught with more difficulty since ascites also disturbs respiration.

Some diseases of the liver cause jaundice, while in other cases enlargement of the liver or a tumor in the portal space can be diagnosed by palpation. Removal of a part of the fluid greatly facilitates this examination in that the abdominal wall is relaxed and the contents of the abdomen are felt far more easily.

**Prognosis.** The prognosis is unfavorable, as a rule, because ascites is generally due to some incurable primary disease. Exceptions to this rule are cases of hydremia and cases of ascites due to excessive feeding with materials rich in water. In such cases a complete change of diet is sufficient to effect a cure, provided it be not too late. A more favorable prognosis can be given in cases of simple ascites in young dogs.

**Treatment.** Recovery promptly follows the removal of a part of the liquid in cases of ascites in young dogs. Exceptionally similar recoveries take place in the horse and ox (Brusasco, Hajnal, Fekete). The ascites occurring in so-called dropsy of the cellular tissues (Zellgewebswassersucht) of cattle is cured by the administration of dry food, provided the case is not too far advanced. Good hay, lucerne and corn should be given.

In practically all other cases treatment is symptomatic and aims at relieving the functional disturbances caused by the collection of the liquid. It is, as a rule, only dogs and other pets that offer the opportunity for treatment, this being rarely undertaken in animals that can be slaughtered or in horses.

The absorption of the transudate is assisted by diuretics, infusion and other preparations of digitalis (see page 121), strophanthin (see page 121), sodium benzoate or citrate of caffeine are valuable. The salts of caffeine may be given (to dogs) in doses of 0.1-2 grams three or four times a day. The following diuretics should also be mentioned; potassium acetate in doses of 0.1-0.2 grams three or four times a day. The infusion of digitalis, juniper juice (1.0-2.0 gm.), squills either as



powder or infusion, diuretin (0.5-1.0 gm. every two hours), agurin (1.0-2.0 gm. daily), theocin (0.6-1.2 gm. per fifty kilos of body weight [Albrecht]). Purgatives are less to be recommended on account of their less certain action and the debility of the animal. Besides the neutral salts, calomel is the drug principally employed. Small doses (0.3-0.4 gm. for dogs) are given, and if necessary combined with jalapin.

Provided that the condition of the heart and lungs permit of its use, injections of pilocarpine produce more favorable results by stimulating diaphoresis and the secretion of large quantities of saliva. The dose that may be employed for dogs is 0.005-0.1 gm. in watery solution and this may be repeated daily. According to Zahns five to ten drops of a 1 per cent solution may be given three times a day. Arecoline may be used in some cases.

Puncture is indicated in cases where the quantity of liquid is more considerable and this may have to be repeated on account of the appearance of symptoms of dyspnea. In less severe cases this treatment yields good results, inasmuch as after the removal of a part of the liquid there is pronounced improvement or even a complete disappearance of the condition for a time. Even repeated tapping is borne moderately well if it be possible to compensate for the loss of albumin by suitable nourishment.

Tapping is practiced at the lowest part of the abdomen with the animal in the standing or if necessary in the prone position. After proper preparation of the seat of operation (shaving and washing of the skin with alcohol or ether) the abdominal wall is punctured with a sterilized trochar of not too wide a bore, or with the needle of a hypodermic syringe. The stiletto is withdrawn and the tap of the canula closed. To the canula is attached a long piece of rubber tubing, the free end of which is put into a clean glass vessel placed on the ground. The cock is then opened and the liquid allowed to run out. If the flow ceases movements are made with the canula in order to displace the piece of mesentery or loop of intestine that is causing the obstruction. Should this prove unsuccessful the rubber tube is removed and a sterile probe is introduced in order to remove any pieces of fibrin that may be sticking in the canula or to displace any organ that may be covering the opening of the canula. When the liquid has been withdrawn the wound in the abdominal wall, which is as a rule covered by the skin which was displaced before the deeper layers were penetrated by the trochar, is sealed either with collodion or with a pledget of wool soaked in pitch. It appears to be advisable to put on a pressure bandage. Dieulafoy's suction pump may be used for the withdrawal of the liquid.

Tapping needs to be carried out with care and as slowly as possible as a too rapid removal of the liquid may lead to rupture of blood vessels, dangerous disturbances of function of the heart or acute cerebral anemia. Should there be a sudden appearance of symptoms of collapse during tapping this must be stopped immediately and injections of ether or camphor given, and if necessary black coffee may be administered. At each subsequent tapping a fresh seat of operation must be selected.

To prevent fresh effusion of liquid, French authors (Presseccq, Sansot) advise intraperitoneal injections of tincture of iodine or Lugol's solution. This treatment ought to give the best results in a primary case, but it is dangerous in that it may cause a rapidly fatal peritonitis. More recently the intraperitoneal injection of a 1 in 5 solution of choral hydrate solution has been advised by Sendrail & Cuillé. Directly a portion of the transudate has been removed from the abdomen a syringe is connected to the rubber tube and the sterilized watery solution is introduced (about 1 gm. per 3 kilograms body weight). As a rule several injections are necessary at increasing intervals. The authors record good results with this treatment.

Omentopexia (Talma's operation in human surgery) is also recommended by



Sendrail & Cuillé. This involves the insertion of a piece of the great omentum measuring about a hand's breadth between the oblique muscles of the abdominal wall, thus establishing a collateral outflow from the portal system into the general circulation. Satisfactory results were obtained in three cases treated in this way.

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## 2. Peritonitis.

**Occurrence.** Peritonitis is, as a rule, a secondary condition and occurs most frequently in the horse, owing to the susceptibility of this species to severe colic of different types. Next to the horse, the ox, and especially the cow, is likely to suffer from peritonitis, puerperal infection and traumatic gastritis being the primary diseased conditions. The disease is far more rare in the other domestic animals and in birds.

**Etiology.** Apart from some cases that are chronic from the outset, the real cause of peritonitis is an infection of some sort. Several varieties of bacteria are capable of setting up the disease. The bacillus bipolaris in cases of hemorrhagic septicemia (see Vol. I) is a well-known cause of peritonitis. In these cases the inflammation is generally slight. The pyogenic bacteria, viz. staphylococci, diplococci and streptococci and the bacillus coli communis which is always present in the intestine, are frequent causes of peritonitis, the latter especially in cases that are associated with disease of the intestine (Borszéký & Genersich, Hensen). The tubercle bacillus, bacillus pyogenes, streptothrix (actinomyces) canis, in some cases the anthrax bacillus, the bacillus of swine erysipelas (Eisenmann) and other organisms may give rise to peritonitis. In fowl cholera the peritonitis is sometimes so prominent a lesion that it may be considered as an independent condition.

Preisz records a case of peritonitis and pericarditis with local necrosis at the seat of inoculation in a horse vaccinated against anthrax. A rounded or short oval bacillus was cultivated from the blood which set up acute inflammation of the serous membranes after subcutaneous inoculation into experimental animals. On the surface of agar the organism formed rounded, flat, bluish, transparent colonies, and after several weeks gelatin cultures showed bluish-white colonies and streaks in the depth of the medium. The bacteria reached the peritoneum from the seat of inoculation by way of the blood stream.

In the exudate from a case of serous peritonitis in a horse Hamburger found streptococci (*Str. peritonitidis equi*). The organism grew on horse serum and in gelatin, agar and broth prepared from horse flesh. Intraperitoneal inoculation of cultures into a horse set up serous peritonitis accompanied by fever.

Injuries to the abdominal wall are very frequent causes of peritonitis (*peritonitis traumatica et operativa*) if the peritoneum be injured, because the peritoneal cavity is laid open

to the entrance of infective material. Violent blows with blunt objects are likely to set up at most a circumscribed peritonitis only. Besides accidental wounds of the abdominal wall, those made in the course of operations play an important part in the production of peritonitis. Among these may be mentioned laparotomy, operation for hernia, castration, etc. Simple puncture carried out with all precautions as to asepsis is far less dangerous. Horses are particularly susceptible to infection in this way and this fact is no doubt associated closely with the high degree of susceptibility that horses, as a rule, show to the pyogenic bacteria.

Injury to any of the abdominal viscera, the contents of which are not germ free, is exceedingly likely to set up peritonitis. In cattle foreign bodies are a very common cause of peritonitis, but cases of this sort are far more rare in other species of animals, although, of course, foreign bodies accidentally swallowed may cause perforation of the stomach or intestine. Included among such cases must be mentioned rupture of the rectum during rectal examination, the administration of a clyster, or copulation. Rupture of the vagina or uterus during dystocia and especially as a result of surgical interference, rupture of the oviduct in birds, rupture of the bladder in cases of retention of urine or perforation during passage of a catheter, rupture of diseased gall bladders, etc., may all be causes of peritonitis. Rupture of the stomach or intestine resulting from ulceration or excessive distension, or more rarely from traumatic causes, sets up peritonitis of the same type (peritonitis perforativa).

Peritonitis may also follow rupture of the other abdominal viscera if these be the subject of a pre-existing bacterial disease. From various causes (strangles, foreign bodies, animal parasites) there may be suppuration of the liver, spleen, prostate, testicle, spermatic cord, and the walls of these abscesses may burst. In cases of puerperal metritis there may be pus in the Fallopian tubes and rupture of the uterus permits of easy infection of the peritoneum. According to Lignières the streptococcus albus and aureus do not rarely occur in a healthy uterus.

In bacterial inflammations of the abdominal organs the inflammatory process may extend to the peritoneum without there being any rupture of a viscus, the infection reaching the peritoneum either directly or by way of the lymph stream. In this connection deep-seated inflammation of the stomach, intestine and interior of the bladder or sexual organs are most dangerous. In severe intestinal disorders the bacteria reach the peritoneum either by multiplying in the diseased wall of the intestine until they reach the serous coat, or they escape in the extravasated serum.

Bongartz frequently observed peritonitis in cows newly brought into cow houses and thought that it was connected with active fermentation processes in the intestine, the products of the fermentation favoring the passage of the bacteria through the intestinal wall.



Pleurisy very rarely sets up peritonitis by extension.

In the group of acute infective diseases known as the hemorrhagic septicemias, peritonitis is comparatively common, but in the specific septicemic diseases, such as anthrax, swine erysipelas, etc., diffuse peritonitis is rarely met with.

Finally, peritonitis may be caused by bacteria carried by the blood stream from lesions in peripheral parts of the body.

Peritonitis associated with gastritis or septicemia has often been observed in sheep and cattle that have been fed on bad turnips.

In Upper Italy, Boccari saw cases of rapidly fatal sero-fibrinous peritonitis in calves from 2 to 4 months old. Organisms resembling Fränkel's diplococcus were found in the exudate. The organism was fatal to mice and rabbits in 24 hours. Material taken from experimental animals was non-pathogenic for calves but the exudate from diseased calves was proved to be pathogenic.

In birds hyphomycetes (*Aspergillus fumigatus*) is sometimes responsible for peritonitis. Lignières & Petit observed an epizootic peritonitis of this nature in turkeys. Gougerot & Caraven saw two cases of peritonitis in young dogs due to the *Sporotrichum Beurmanni*.

In very rare cases microorganisms cause peritonitis without there being any injury to the abdominal wall or disease of any sort in the animal at all (primary peritonitis).

Among the predisposing causes of primary, and in some cases of secondary, peritonitis may be mentioned cold (rheumatic peritonitis), debility, violent blows on the abdominal wall with blunt objects, etc.

Chemical substances, such as bacterial toxins and materials retained in the blood as a result of nephritis, are very rarely responsible in themselves for peritonitis. Sterile bile, urine and other liquids do not cause the disease. Occasionally animal parasites are responsible for peritonitis, this having been observed in carnivora by Baillet, Railliet, Labat and Cadéac. Very occasionally cases have occurred in cattle and pigs as a result of a severe invasion of *Cysticercus tenuicollis* or the liver fluke.

Chronic peritonitis is generally due to chronic or repeated attacks of sub-acute inflammation in neighboring organs, whereas external or internal injuries, as a rule, set up a localized peritonitis which results in adhesions of the peritoneal surfaces. Chronic peritonitis is seen in tuberculosis, glanders and accompanying carcinoma, sarcoma and other neoplasms of the serous membrane. It may also occur in animals that are in a debilitated condition and as a result of ascites. The frequently occurring cases in horses associated with the formation of false membranes are due to an antecedent invasion by larval sclerostomes (Glage. See page 490).

Fuchs observed peritonitis with adhesions in the neighborhood of the stomach of cattle that had been fed on turnips stored in trenches, and believed that a pathogenic mould grew in the fermenting roots which produced gastritis and subsequent peritonitis.



**Pathogenesis.** Under the influence of the cause of the inflammation the blood vessels of the peritoneum dilate and the endothelial cells are shed at places and there is an immediate escape of serum and red blood corpuscles into the peritoneum. Owing to the richness of the peritoneum in nerves the pain due to the inflammation is continuous and severe and is increased by pressure. As the surface presented by the peritoneum to the absorption of bacteria and their products is very large, these pass into the blood in large quantities from the very outset and produce general disturbances, the extent depending upon the area involved in the inflammatory process.

If in addition to the cause of the inflammation bacteria products are poured into the peritoneum in large quantities, as is the case in extensive rupture of an organ, absorption of the toxin may cause death before peritonitis has had time fully to develop. Acute inflammation decreases peristalsis and later suppresses it entirely. This is due to the serous infiltration of the muscular tissue and still more to reflex action.

The introduction of bacteria into the peritoneum by no means always leads to peritonitis. Experiments have shown that pathogenic bacteria may be quickly absorbed by the healthy peritoneum (Grawitz, Pawlowsky and others), and that they can only exert their pathogenic effect if they are introduced in large numbers or if they cannot be easily absorbed, as when the peritoneum is injured in some way, either traumatically or chemically, or owing to some disturbance of circulation. Tavel and Lanz go so far as to state that bacteria produce no effect save in an already diseased peritoneum and that therefore there is no such thing as primary peritonitis.

**Anatomical Changes.** In the early stages the peritoneum is markedly congested, dull and opaque, and is frequently studded with punctiform hemorrhages. Within the next twenty-four hours there form thin filmy fibrinous membranes which are easily detached. These may subsequently measure several millimeters in thickness, and while still remaining soft and easily torn, cement opposed layers of the peritoneum together. There is a simultaneous accumulation in the peritoneum of a turbid serous fluid containing flocculi of fibrin. In the horse there may be as much as forty liters of such exudate and in cattle the amount may be as much as 100 liters. In other cases the exudate is purulent. In cases of rupture of the stomach or intestine the exudate is mixed with food or intestinal contents and in cases where the perforation is effected slowly the exudate is sanious. In any case the exudate may contain a larger or smaller proportion of blood. Chronic peritonitis is characterized by the formation of connective tissue. The encapsuled abscesses in cattle and pigs due to infection with the bacillus pyogenes are produced in this way. Apart from this, acute fibrinous peritonitis usually becomes chronic with the development of connective tissue under the fibrinous membranes. The peritoneum becomes thickened and the surface of the organs is covered with thick layers of connective tissue. Both these forms are seen very often in the



liver and spleen of the horse. Chronic peritonitis is very often circumscribed in extent, especially on the serous membranes of the liver and spleen, seats of puncture of the rumen or cecum, and in the pelvis around the bladder and sexual organs. In cattle, and still more frequently in pigs, chronic peritonitis leads to the formation of bony plates close to the wound made for ovariectomy (Gurlt, Johne, Deland).

**Symptoms.** In view of the fact that peritonitis is generally a secondary condition, its symptoms are preceded by those of the primary disease, but the latter give way to those of peritonitis either gradually or suddenly, according to whether the infection of the peritoneum is severe or not. The primary disease is, as a rule, some severe disease of the stomach, intestine or genital organs. If the peritonitis is due to an acute septicemic disease its symptoms are more or less obscured by those of the primary disease, but in other cases the symptoms of peritonitis are very obvious.

In cases of **acute diffuse peritonitis** abdominal pain is the most pronounced and usually the most persistent symptom. Animals show evidence of pain in a variety of ways, groaning, whining, bellowing, grinding the teeth, looking round at the body, switching their tails, etc. They avoid all sudden movements, stand with their feet close under the body, either arch or hollow their backs, and in the latter case hold their heads and necks drawn back. Horses often try to lie down, but, as a rule, do not get beyond the attempt; should they succeed, however, they exercise great care in carrying out the movement, and in some cases lie on their backs. Cattle, as a rule, remain standing and only lie down just before death. Small animals lie in a crouching attitude, and their countenances are often expressive of great pain. The abdominal wall may be painful all over, or only in parts, and the animals try to avoid all pressure on it. Motion is stiff and only short steps are taken, any deviation from the straight line being avoided. If the inflammation is localized in the anterior part of the abdomen, going uphill is especially painful.

The abdominal pain and the susceptibility to pressure of the abdominal wall vary from individual to individual, but to a still more marked degree in the various species. In the horse the symptoms of pain are principally subjective, and they are never completely absent; whereas susceptibility to pressure on the abdominal wall is very seldom demonstrable, owing to its natural and in this case increased tenseness. In small animals the opposite holds good and in cattle both subjective and objective symptoms are less clearly shown and may be absent altogether.

The abdomen remains normal in size in the early stages and may be even less than normal in circumference owing to reflex

contraction of the muscles. Increased distension may best be appreciated by palpation, and fibrillar muscular tremblings can frequently be felt.

Friction sounds, due to the movement of roughened layers of peritoneum on each other, can be heard only rarely. This is most frequently observed in cattle and in the left hypochondriac region if the inflammation involves the diaphragm (foreign body), and in the right flank in cases of metro-peritonitis (Detroye). The roughness and painfulness of the peritoneum can easily be discovered by rectal examination, and in many cases of rupture of the stomach or intestine, particles of food can be found in the peritoneum.

Where large quantities of fluid are present the flanks are depressed and the abdomen gradually becomes wider in its lower part. In such cases an area of dulness limited above by a horizontal line can be made out.

Neighboring organs are frequently affected in sympathy. In carnivora and swine there is often vomiting and intestinal contents may be found in the vomit. This symptom results from irritation of the peritoneum, especially over the stomach wall and has been observed in exceptional cases in the horse (Röll). On the other hand, extension of the inflammation to the diaphragmatic layer is sometimes followed by persistent sobbing (Anacker). In cattle Otto noticed peculiar rolling sounds following every movement of the rumen.

In the early stages of the disease there is diarrhea, and active intestinal murmurs can be heard, provided the primary disease is not one in which peristalsis is suppressed. Very soon and in some cases from the outset, constipation sets in which is of a very persistent character. In the later stages there is general meteorism in all animals, and in cattle distension of the rumen is a prominent symptom. Animals strain frequently and while doing so, groan with pain and show other signs of pain in the intervals.

Frequent urination with pressure of urine and tenesmus of the bladder are sometimes observed. Exceptionally there is retention of the urine which may be due to paralysis of the muscular tissue of the bladder following on edematous infiltration, or to reflex contraction of the sphincter.

Respiration is costal and accelerated, all movement of the diaphragm and the abdominal organs being avoided as far as possible. With the outpouring of the exudate and the meteorism the difficulty of respiration becomes more pronounced.

In acute peritonitis in cattle Cuny observed a bilateral flow of tears which soon became converted into a purulent discharge, associated with redness and swelling of the conjunctiva and opacity of the cornea. He also noticed a watery nasal discharge which in the later stages became mucoid.

In all cases there are symptoms of general disturbance.

Fever is very rarely absent and is, as a rule, high. The



fever takes no typical course (Fig. 77); it is sometimes continuous and at others remittent or intermittent. In carnivora and more rarely in other animals the temperature at the onset and even up to the end may be subnormal in cases of perforative peritonitis. The pulse, which is quite independent of the temperature, is without exception accelerated from the outset and may be twice as rapid as normal or even faster. In the early stages it may be rather tense, but soon becomes very weak and may be nearly if not quite imperceptible, probably on account of the serious fall in blood pressure. To this are due the sudden cooling of the extremities (especially

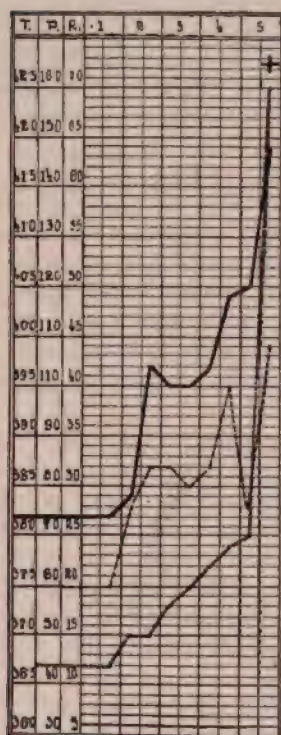


Fig. 77. Temperature chart of a case of secondary diffuse peritonitis in a horse due to injury of the interior of the intestine by a calculus.

the ischial region and root of the tail [Otto]), the cyanosis and pallor of the mucous membranes, and also, at least to some extent, the languor and prostration. The animals are quite dull and take notice of nothing, or they lie down and are very difficult to move (cattle and small animals in particular). There is, as a rule, a complete absence of appetite.

In severe septic conditions (septic metritis, rupture of an abscess) peritonitis develops rapidly with the symptoms of pyemia or septicemia, there being high fever and languor with the early onset of diarrhea, but no symptoms of pain.

In cases of rupture of the stomach or intestine there is a sudden depression, the extremities are very cold, the pulse is thready and very rapid and the body is bathed in cold sweat. In such cases the excessive tenseness of the abdominal wall, the suddenly developing distension followed by a rise of temperature, the rough condition of the peritoneum which is sometimes discoverable by rectal examination, and the complete collapse indicate peritonitis.

In cases of **circumscribed acute peritonitis** the pain is generally less severe and localized. The general symptoms are less pronounced and the principal

symptoms observed are due to disease in neighboring organs. Such localized peritonitis may lead to the formation of adhesions and the encapsulation of the exudate, but there is always the danger that under certain circumstances there may be a general peritonitis spread from such places.

The symptoms of **chronic peritonitis** are, as a rule, so slight and indefinite that it is very rare that a definite diagnosis can be made. Gradual wasting associated with variable temperature,

digestive disorders with persistent diarrhea suggest chronic intestinal catarrh, while in the horse occasional attacks of colic which last only for a brief time have no diagnostic importance. Diagnosis is generally possible only if in addition to the above mentioned symptoms there is localized sensibility to pressure of the abdominal wall, if there is an accumulation of fluid within a short time, which proves to be an exudate on test puncture. In cases of chronic peritonitis with effusion there is sometimes subcutaneous edema of the abdominal wall.

Localized chronic peritonitis frequently causes disturbance of other organs (loss of tone of the rumen in cattle, stenosis of the intestine). The exact nature of these derangements can be determined in rare cases only by rectal examination or palpation of the abdomen.

**Course.** Acute peritonitis is sometimes very rapid in its development and may cause death in the first stage of the disease and even within a few hours. This is particularly the case where there is an escape of stomach or intestinal contents or of pus into the peritoneal cavity. It is seen only exceptionally in cases of puerperal infection. In such cases the local symptoms are less pronounced and the symptoms shown are those of general sepsis associated with high fever, rigors, occasionally a subnormal temperature, weak and rapid pulse and great prostration, or of a putrid intoxication. In the great majority of cases the symptoms develop slowly, death taking place from the fourth to fourteenth day. Sometimes there is a marked fall in temperature before death and there is usually diarrhea. In other cases the disease becomes chronic and may last for months or even years. The same is true for cases that are chronic from the outset.

**Diagnosis.** The moderately rapid development of the disease, fever, tenderness of the abdomen to pressure, constipation and tympanites, acceleration of the pulse from the commencement and gradual weakening of the heart beat form a fairly characteristic picture. Diagnosis is more certain in cases where, owing to a previous examination of the animal or a reliable history, a primary disease is known to be in existence. Otherwise it can only be based on the discovery of a roughened condition of the peritoneum, adhesions between various organs or the presence of a fluid exudate in the peritoneal cavity.

In cases of peritonitis due to displacement of stomach or intestine the exudate obtained by tapping is at first generally clear and reddish in color, but as the amount of liquid increases, owing to disturbance of the circulation, and the peritonitis becomes more severe the liquid becomes turbid.

In the horse the disease is likely to be confounded with gastric and intestinal colic in which the pulse is rapid and weak from the outset, or the animals are inclined to be persistently restless. In gastro-enteritis there is diarrhea either through-

out or at any rate for a considerable time and loud intestinal murmurs, but rectal examination reveals no readiness of the peritoneum nor is it painful. Other kinds of colic can be differentiated by the facts that in them there is no rise of temperature for some time at least, and that the animals are restless, and rectal examination affords in many cases much valuable information. All forms of colic may lead to peritonitis in the later stages, and especially those due to thrombosis and to displacement of organs. In the dog cases of volvulus of the stomach may be confounded with peritonitis (see page 317). As a rule chronic peritonitis can only be diagnosed when the presence of fluid can be recognized in the peritoneum, or there is known to have been an antecedent acute attack. At most, limited areas of inflammation of the peritoneum will be found where there have been some adhesions formed (see stenosis of the intestine). The differential diagnosis of chronic serous peritonitis and ascites has already been dealt with (see page 568).

**Prognosis.** Acute diffuse peritonitis must be considered as a very dangerous condition and the prognosis is very unfavorable when the inflammation is due to perforation of the stomach or intestine, or to the escape of pus into the peritoneum. Less extensive localized fibrinous inflammation is commonly unassociated with any danger, and is in many cases not recognized during life. Prognosis is more favorable in cases due to injury from the outside, because with suitable treatment the disease may be kept localized and a cure effected. Extensive chronic inflammation leads to adhesions between the abdominal wall and some of the viscera, especially intestine, and consequently digestive disorders of a permanent nature are produced.

**Treatment.** In cases where the peritonitis is due to some wound, such as castration, antiseptic treatment immediately after the first symptoms make their appearance will frequently arrest the extension of the disease and limit it to the immediate neighborhood of the wound. Surgical intervention may be followed by good results in cases due to encapsuled abscesses, such as are produced in cattle by foreign bodies. Finally the good results obtained in human surgery indicate that in certain cases, especially in carnivora, cures might be effected by surgical intervention, such as laparotomy, suturing of the wound in the intestine, resection of the intestine, washing out the peritoneum with sterile salt solution at body temperature, or with weak solutions of salicylic or boric acid. Baldoni cured a dog in seven days by flushing the peritoneum. To decrease the absorptive power of the peritoneum Wilms advises in human surgery the intraperitoneal injection of 130 cc. of oil of camphor. Attention should next be paid to decreasing the movements of the intestine to avoid as far as possible the dissemination of the infective material by the peristaltic action. This object is best



attained by the administration of opium in doses of 5-10 gm. for horses and cattle, 1-3 gm. for small herbivora and 0.1-0.3 for dogs. Morphine hydrochloride may also be given in doses of 0.3-0.4 gm. or 0.02-0.1 gm. subcutaneously once or twice daily. Provided the primary disease permits of the administration of food this should be easily digested and if possible in fluid form (gruel, soup, milk). The inflammation should be combatted with cold compresses. These may be made by binding linen bandages around the abdomen and soaking them in cold water, or in small animals an ice bag may be placed on the abdomen. After the disappearance of pain and the acute symptoms, fomentations or rubbing with turpentine should be resorted to (Priessnitz). Gray mercurial ointment may be rubbed into the inner surface of the thigh and the abdominal wall repeatedly so long as there is no salivation. For horses 5 gm. and for dogs 1 gm. may be used daily. Mild purgatives, such as castor oil (horses 250-500 gm. and dogs 15-30 gm.) and calomel (dogs 0.03-0.05 gm.) are indicated for the constipation during the early stages. Lukewarm clysters are advantageous. Cattle should be given, at intervals of two to three hours, water, barley water, oatmeal or linseed tea to prevent the drying up of the food. In cases of enteritis of some standing disinfectants may be tried (creolin, lysol, resorcin, naphthol, etc.; see page 332).

Tapping is indicated for the removal of the fluid and this may be repeated as often as necessary. In some cases puncture of the stomach or intestine may be necessary. In chronic peritonitis attempts may be made to promote the absorption of the exudate by Priessnitz' poultices and massage, potassium iodide (5-10 gm. for large animals or 0.3-0.5 gm. for small animals daily) and diuretics. Any derangements in other organs must be treated.

**Literature.** Baldoni, Clin. Vet., 1900, 28.—Boccalari, Pr. Vet., 1898, 1.—Bongartz, D. t. W., 1897, 392. Cuny, Journ. Vét., 1908, 647.—v. d. Eeckhout, Ann., 1906, 383.—Eggmann, Schw. A., 1892, XXXIV, 151.—Eisenmann, Monh., 1906, XVII, 97.—Emmerich, A. f. Tk., 1899, XXV, 222.—Glage, D. t. W., 1903, 442; Z. f. Infkr., 1906, I, 341.—Hamburger, Chl. f. Bakt., 1896, XIX, 882.—Knoll, B. t. W., 1899, 146.—Lignières & Petit, Rec., 1898, 145.—Moussu, Rec., 1903, 549; Rev. Gén., 1903, II, 9, 593.—Otto, S. B., 1900, 49.—Preisz, Vet., 1893, 509.—Pr. Mil. Vb., 1899-1908.—Röder, S. B., 1893, 121.—Scheuerlen & Buhl, B. t. W., 1901, 369.—Wilhelm, S. B., 1892, 102.—Wohlmuth, Ö. M., 1900, 263.

### 3. Tumors of the Peritoneum.

**Occurrence.** In addition to tuberculosis and actinomycosis true neoplasms may occur in the peritoneum primarily and metastatically. Diffuse carcinoma and more rarely sarcoma (melanotic) of the peritoneum are seen sometimes. The membrane is beset with neoplasms of various sizes and the connective tissue layer appears to be thickened, while the neighboring viscera may be more or less compressed or atrophied. Fibromata, lipomata and myxomata, and very exceptionally angio-

**mata** occur in the peritoneum. They are practically always attached either to the omentum or mesentery and are, as a rule, solitary. The emphysematous cysts of the mesentery frequently found in the pig and due according to Jäger (A. f. Tk., 1906, XXXII, 410) to a bacillus of the colon group, the *bacillus coli lymphaticum ærogenes*, are of no importance from a clinical point of view. The same condition was found by Günther in a fowl.

**Symptoms.** As a rule neoplasms cause only disturbances of a general nature and possibly also abdominal pain, especially carcinoma and sarcoma. There is gradual wasting until a condition of extreme debility is produced. In certain cases the symptoms present indicate stenosis or occlusion of the intestine, the cause of which can only be determined by the positive result of palpation or rectal examination. The symptoms of chronic peritonitis or ascites are more valuable in this respect. Chylous ascites (see page 565) in particular raises the suspicion of carcinoma of the peritoneum. This suspicion is strengthened if a primary growth can be discovered in any other organ, such as the testicle, udder or prostate.

**Treatment.** In large animals and in cases where the neoplasm is situated in a part of the peritoneum accessible to operation, surgical intervention is the only treatment possible. No good results are to be expected if the growth be of a malignant nature.

#### 4. Animal Parasites Found in the Peritoneum.

The animal parasites which occur either free in the peritoneal cavity or in the peritoneum are of very little importance from a clinical point of view. It is only exceptionally that they cause any disturbance of health and their presence can scarcely ever be discovered during life.

The *Filaria papillosa* occurs very frequently in the peritoneal cavity in the horse but it is quite harmless. Occasionally echinococci are found. In a case recorded by Toutey a cyst hanging by a pedicle was the cause of a twist. *Cysticereus fistularis* Rudolph, Reckleben and the larvæ of *sclerostomes* also occur. The latter may be either encapsulated in connective tissue or in hemorrhagic foci in the subperitoneal tissue, or there may be fully developed specimens free Kitt.

In **ruminants** the *Cysticereus tenuicollis*, see page 468, occurs with great frequency and may cause acute peritonitis if the infestation be excessive. The liver-fluke (Morot) and the *Filaria labiopapillosa* (Alessandrini) occur very rarely.

In the **pig** echinococci, *Cysticereus tenuicollis* and in America the thread-like *Stephanurus dentatus* are found.

In **carnivora** the following parasites may be found, echinococci (Railliet, Reinmann and Pécard, who in one case, found several thousand, some free and some attached to the peritoneum, *Pleurocercoides Bailleti* (found by Baillet and Cadéac each in a cat and by Labat and

Railliet in dogs, sometimes in association with ascites, and in one case the liquid obtained by tapping contained large numbers of cysts), and finally the *Pentastomum denticulatum* (found by Roche-Fontaine in large numbers enclosed in cysts).

In the *Revue Vétérinaire* (1906, 141), there was described by Sendrail & Cuillé under the term "*Hydrops ascites parasitarius*," a disease of the dog caused by worms of unknown origin. The disease occurs occasionally in the neighborhood of Toulouse, one or two cases coming under observation in the clinic every year. The disease differs from ascites clinically only in that the serous liquid in the peritoneum contains numerous cyst-like bodies which vary in size from a pin's head to a pea, are of variable shape and easily crushed, and which on standing separate out into a pasty mass from the otherwise clear liquid. They are motile. The disease may be transmitted experimentally by the introduction of living cysts into the peritoneal cavity of dogs.

According to Sendrail & Cuillé it is an abnormal form of the cyst of the *Mesocystoides lineatus* which is frequently found in the intestine of dogs in the neighborhood of Toulouse. The numerous cases observed in the past by Neumann and classed as echinococcosis should possibly have been described as this disease.



# Diseases of the Nervous System

## SECTION I.

### DISEASES OF THE BRAIN.

#### General Symptomatology of Diseases of the Brain.

Organic diseases of the brain and its membranes are characterized partly by symptoms of a general nature and partly by focal symptoms. The general symptoms are caused either directly by diffuse disease of the cortex of the brain associated with an increase of intracranial pressure, or by an increase of pressure alone. On the other hand localized symptoms are, as a rule, associated with disease of definite portions of the brain connected with certain functions. These two groups of symptoms may be present simultaneously, or either may be present separately.

#### (a) General Cerebral Symptoms.

The most constant symptoms of disease of the brain are various forms of disturbance of consciousness. There may be numbness, dulness, stupor to varying degrees, or complete coma, and they are generally associated with lessened sensibility. The symptoms may set in suddenly as in cases due to hemorrhage, concussion or embolism. In other cases the onset is slower as in acute encephalitis and meningitis, hyperemia and anemia of the brain. Finally the symptoms may take weeks, months or even years to develop as in chronic hydrocephalus, tumors, parasites, encephalitis due to distemper in dogs. Not rarely during the course of diseases of the brain there are remissions of the symptoms. In cases where the disease is less extensive or is localized in the neighborhood of the medulla oblongata there may be a complete absence of them, and cases have been observed in which in spite of a considerable but slowly progressive reduction in the size of the cranial cavity the animals have shown no symptoms whatever.

In acute diseases of the brain, excitement is generally observed, a condition of disturbed perception with simultaneous acceleration of the motor reaction, which, as a rule, is of short

duration and after its disappearance the particular disturbances are aggravated.

Giddiness is a frequent symptom of disease of the brain and usually results from a temporary loss of consciousness.

Abnormal movements are seen in both acute and chronic conditions. They may be associated with loss of consciousness and may affect either the whole body or only a part in the form of tonic-clonic spasms (eclamptoid or epileptiform cases), and also there may be forced movements and ataxia.

Both the frequency and rhythm of the respiration may be altered. In complete loss of consciousness and in deep stupor the respirations are deep and their frequency diminished. In severe cerebral diseases respiration of the Cheyne-Stokes type is not rarely observed. In this type of breathing after a number of respirations of different lengths there is a pause which may last for several seconds to be followed by respirations which at first are superficial and which gradually become deeper and even dyspneic. This is followed by another pause and the respirations become gradually shallow again. In other cases of encephalitis and meningitis respiration of the Biot type is observed. In this type a number of normal respirations are followed by a long pause. Another type of respiration is also observed in which a long pause follows a series of respirations which are at first violent and gradually decrease. As a general rule any serious change in the type of respiration is an unfavorable point in connection with prognosis.

Changes in the rapidity of the pulse are frequently observed. In diseases in which there is an increase of intracranial pressure stimulation of the vagus slows the pulse rate, but should there be any other factor in operation, such as fever, excitement, heart weakness, which has an accelerating effect on the pulse, stimulation of the vagus is without effect. In some cases of serious disease of the brain there is acceleration of the pulse which is of the utmost importance, as there are no other factors tending to produce this in operation. In all these cases the pulse may also be irregular. Considerable variations of the number and rhythm of the pulse are observed in cases of meningitis.

Vomiting is seen frequently in carnivora and swine in cases of diseases of the brain, and especially in acute meningitis and is due either to direct stimulation of the vomiting center or of the sensory nerve fibers of the meninges. The vomiting center in the other animals is far less sensitive to stimulation and, as a rule, they do not vomit in cerebral diseases.

According to some authors (Esberg, Lustig) venous congestion of the papilla of the optic nerve is caused by increased intracranial pressure and especially in cases of chronic dropsy of the ventricles. This should not be of very rare occurrence in animals, but the majority of veterinary authors have not ob-

served this. Such symptoms would be most likely to arise in cases of tumor-formation in the brain.

Exaggeration of the tendon reflexes has been observed especially in cases of chronic encephalitis owing to the cutting out of the inhibitory effect of the cerebral cortex. Contrary to what is the case in man the exaggeration of the tendon reflexes may be due to the cutting out of the cerebellum. A diminution of the reflexes and especially of the skin reflexes is often seen in rapidly progressive acute encephalitis owing to the increased inhibition of the cortex. In complete loss of consciousness the skin and pupillary reflexes are entirely lost; the tendon reflexes are often entirely destroyed and the pupils do not react to light. On the other hand, the pupils appear very contracted and react only very slightly or not at all to light in simple cases of increased intracranial pressure, although there is no loss of consciousness. The alteration of the tendon reflex and the reaction of the pupil may be observed as local symptoms.

The reflex test gives reliable results in animals. One distinguishes between skin and tendon reflexes. The skin reflex is best tested by pricking the skin with a needle or some other instrument, or often by the use of the percussion hammer, and the tendon reflexes may also be tested with the percussion hammer or some other hard object.

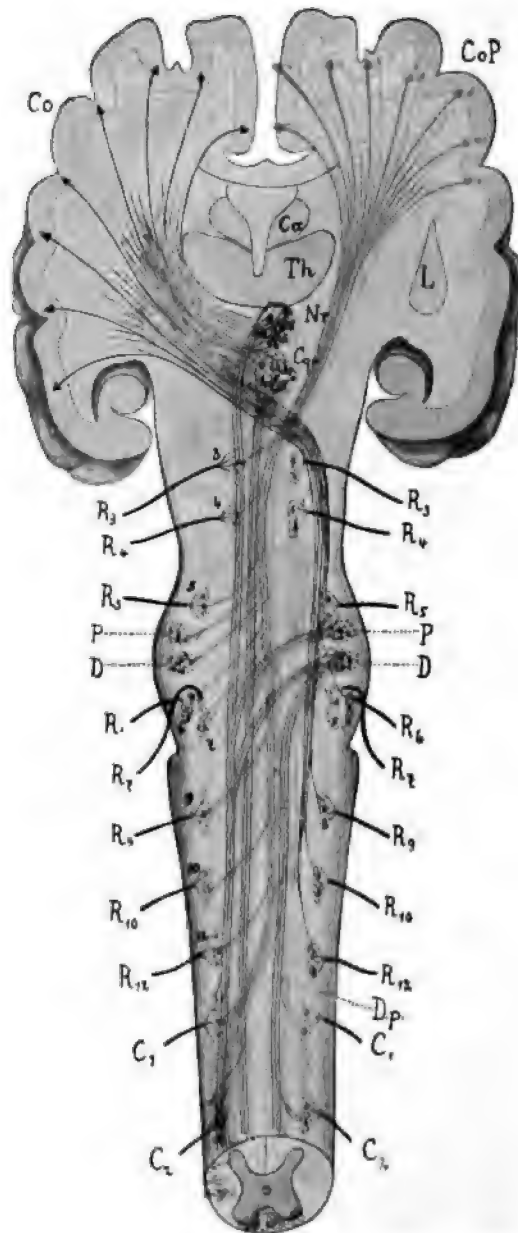
The following skin reflexes may be tested. The reflex at the withers, a twitch of the cutaneous muscle following stimulation of the skin over the withers. In a similar way the skin reflexes over the shoulder, abdomen, sternum, and flank may be tested. The latter test may be applied in all the domesticated animals, the first two to the horse and ox, while the withers reflex as a rule is absent in the ox. The gluteal reflex is a muscular twitch due to a pin prick in the neighborhood of the hip joint. The cremaster reflex may be tested by stimulation of the skin over the inner condyle of the femur, the testicle on the same side being raised. In many stallions and in dogs there is a scrotal reflex, the scrotal skin becoming wrinkled when it is stimulated or when cold water is poured on it. Stimulation of the skin of the perineum causes sinking of the croup, elevation of the tail, contraction of the sphincter ani, and in many cases contraction of the muscles of the croup. The contraction or spasm of the sphincter ani externus which is produced by stimulating the skin of the orifice, or by the introduction of some solid body into the rectum is described by some as the anus reflex. The hock reflex is a contraction of the distal end of the biceps femoris and the semitendinosus induced by percussion of the tuber of the os calcis. Reflexes of the sole are normally seen in carnivora only and in the hind feet. They may be produced by touching the skin of the sole and are evidenced by spasmodic volar flexion of the toes.

Schmidt observed spasmodic contractions of the muscles in the upper parts of the legs set up reflexly in the following ways: pressure or percussion of the hoof (pedal reflex), sudden pressure on the upper third of the pastern (pastern reflex), on the inner side of the upper part of the metacarpus, and on the flexor tendons. These reflexes must not be confused with withdrawal of the limb owing to pain.

The skin reflexes include reflexes of the mucous membranes which are in some instances very complicated, as for example the reflexes of swallowing, coughing, and the bladder reflexes. The eye and conjunctival reflexes are simple. The pupillary reflex is similar. This can be shown in animals in a satisfactory manner only by stimulation by means of light. The pupil becomes narrower when light falls on it if the transmitting media of the eye are uninjured.

Among the tendon reflexes the patellar reflex is of considerable importance, the arc being formed by the femoral nerve and its center. This reflex is not constant in animals in the standing position. The animals must be placed on one side and the upper hind leg is flexed to as great an extent as possible at the stifle; for this help is necessary. The flexed leg being supported with the hand under the hock, a smart blow is struck on median ligament of the patella causing extension of the stifle joint in the backward direction. One must either wait until the animal has ceased voluntary movements of the muscles attached to the patella or adopt some means of distracting its attention from the legs. Under normal conditions





**Fig. 78.** Diagram of the course of the motor paths of the domesticated animals. CoP. Cerebral cortex with the origin of the pyramidal tract shown in red. Co. Cerebral cortex showing the diffuse distribution of the points of origin of the principal cortical motor paths (black). Ca. Nucleus caudatus. Th. Optic thalamus. L. Nucleus lentiformis. Nr. Red nucleus. Cqu. Corp. quadrigemina. P. Pons Varolii and D. Deiter's nucleus with the subcortical motor centers for the principal subcortical paths (blue). 3-7, 9-10, 12 nuclei, R 3-7, R 9-10, R 12 roots of the corresponding cranial nerves. Dp. Decussation of the pyramids in the medulla oblongata. C1-2 roots of the I and II cervical motor nerves.

the reflex is most active in the dog, cat and sheep and least in the ox, the horse occupying an intermediate position. It is as a rule more pronounced in young animals. Reflexes of the anconeus or gastrocnemius can scarcely ever be obtained by striking their tendons under normal conditions.

In pathological conditions reflexes may be either exaggerated, diminished or entirely absent. The exaggeration of the reflex is recognized by spasms which are more extensive and follow each other with greater rapidity. The opposite is the case when there is a diminution of the reflex, but great care must be exercised in determining them because voluntary movements of the muscles may prevent the occurrence of twitches owing to the fact that the animals have to be kept still for a considerable length of time during the investigations.

Disturbances of nutrition are observed, especially in the later stages or in diseases of the brain of long standing. These are for most part due to an insufficient quantity of food being taken owing to severe disturbances of consciousness.

The diagnostic importance of general brain symptoms is that they indicate either a direct or indirect disease of the whole brain, but they are of no value in localization of the disease. It should not be forgotten that general symptoms of a moderate nature may be due to functional disturbances of the brain, such as occur in acute infectious diseases and such as ordinarily occur in severe internal diseases.

#### (b) Focal Symptoms.

The comparative rarity of localized nervous diseases in the domesticated animals and the primitive methods of investigation of the nervous system of animals adopted in earlier times sufficiently explain the fact that our knowledge regarding the localization of diseases of the brain and focal symptoms is somewhat defective. A brief outline of the focal symptoms based upon cases of disease that have been published more recently and upon the animal experiments carried out by various authors appears to be justified.

As a general rule focal symptoms cause motor and sensory disturbances. Motor disturbances are generally the more easily observed and are evidenced either by paralysis or abnormal movements.

**Motor paths** (see fig. 78). Experimentation has shown that there are great differences between the motor paths and centers in man and animals, and that the differences become more pronounced the lower the animal is in the scale of development. The peripheral motor path, the oldest in the vertebrata, begins at the nerve cells in the anterior horn of the gray matter in the spinal cord or in the nuclei of the cranial nerves and terminates in an end plate in a muscle fiber. The cells of origin of this path are connected with the central motor path. In the first place they are connected with the subcortical or principal centers by means of the extrapyramidal or principal paths; further, with Monakow's fasciculus between the nucleus ruber (Nr) and the lateral column of the spinal cord on the opposite side, by a fasciculus connecting the corpora quadrigemina (Cqu) with the ventral column of the opposite side, paths from the pons (P) which pass partly to the lateral column on the same and partly to the lateral columns on the opposite side, and finally a path connecting Deiter's nucleus with the ventral column on the same side. The subcortical motor centers are connected by nerve paths with the motor cells scattered through the cortex and arranged around the sensory centers. These paths, after arising in the cortex converge towards the capsula interna and then crossing over pass to the subcortical centers of the opposite side.

In the mammals the pyramidal tract arises within a definite area (the psychomotor zone), which in the cases of the carnivora is accurately known. It converges towards the white matter of the hemisphere and occupies a narrow space in the internal capsule placed laterally to the optic thalamus. From here it passes to the pons and medulla where either the whole of it or the greater part passes to

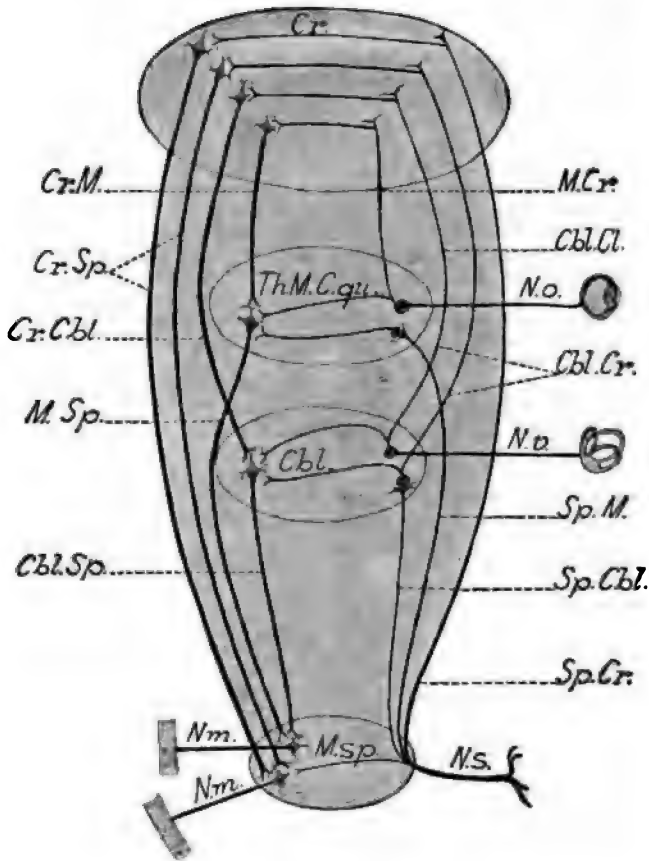


Fig. 79. Diagram of the system of coordination. The sensory parts are on the right, the motor parts on the left and the centers concerned in the middle. Cr. cerebrum. Th. M. C. qu. optic thalamus, methalamus and corpora quadrigemina. Cbl. cerebellum. M. sp. spinal cord. Stimuli from the skin, muscles, tendons and joints pass either to the spinal cord by way of the sensory roots or to the medulla by way of the sensory root of the trigeminus (not shown in the figure). Some are connected by the reflex collaterals directly with the motor cells in the cord and medulla and others through Sp. Cbl. to the cerebellum, Sp. M. in the neighborhood of the thalamus through Sp. Cr. to the cerebrum and then to the motor cells in these centers. The motor impulses produced pass from the cortex by the motor path Cr. Sp., from the thalamus by M. Sp. and from the cerebellum by Cbl. Sp. to the motor cells in the cord and medulla. The cerebellum also receives sensory impulses from the semicircular canals by way of the free vestibular nerve (N. v.), the thalamus and corp. quad. from the organs of vision by way of the optic nerve (N. o.) and these are conveyed to the motor cells in the central organs and cerebrum by the paths Cbl. Cr. and M. Cr. (Adapted from O. Forster).

the other side to be continued in the lateral column of the spinal cord. The fibers composing this nerve path accompany the peripheral motor path, both in the brain and in the spinal cord, so that a connection is established between the cortex of the brain and the peripheral motor nerves without touching the subcortical centers. In



the ungulates the pyramidal tract is very poorly developed and can be traced as far as the 4th cervical vertebra only (Dexler and Margulies). In the carnivora the tract is more fully developed and can be traced as far as the sacrum, but it is less distinct than in man.

In birds there are no direct motor tracts connecting the brain and spinal cord, the whole central motor path being in connection with the subcortical centers. It is only in the parrot that a nerve path comparable to the pyramidal tract occurs (v. Kalischer).

The cells from which the peripheral motor paths arise are collected into groups of varying size, having a common function, and forming numerous reflex centers both in the brain and spinal cord, stimulation setting in action one or more muscles, or under certain circumstances, whole groups of muscles on one or both sides of the body in definite sequence. The complex reflex mechanisms which permit the correlation of movements of certain parts of the body with movements of other parts, such as running, walking, etc., are controlled by the subcortical centers. With development the subcortical centers encroaches upon the sphere of action of the cortex, but apart from this other exactly similar movement can be carried out without calling into play the reflex mechanism, the brain cortex taking part in numerous reflex mechanisms to the exclusion of the subcortical centers. This is rendered possible by the pyramidal tract which in the ungulates controls the head and neck, and in carnivora the limbs also. The lower the position occupied by an animal in the scale of development the greater the independence of the individual elements of the motor paths on the opposite sides.

From the foregoing it is clear that the pyramidal and the extrapyramidal tracts can convey the so-called voluntary impulses of the cortex and be effective on both sides of the body. As all these paths and centers are disposed over a wide area of brain, and as the subcortical centers are somewhat independent of the cortex, lesions capable of causing total central paralysis in animals must be very extensive and more so than those producing the same effect in man.

In the coordination and regulation of movements several portions of the nervous system take part (see fig. 79).

Two principal portions control coordination and regulation; the sensory paths which inform the cells of the central nervous system regarding the individual muscle functions, the position of the whole or of some part of the body, and the motor paths which convey stimuli to the muscles depending upon the most varied sensory impressions in the nerve cells. The component parts of the centripetal path are not all of equal value as regards coordination. In certain muscular functions the special sensory paths are involved, in others the vestibular or the optic paths; in the equilibration of the whole or of parts of the body the paths leading to the cerebellum or even the cerebellum itself may be concerned. Each part of the coordinating system is functional up to a certain point. As the various components are not of exactly equal value they are able, so to speak, to hand over the excess to the opposite side. This is particularly the case with regard to the organ of vision and consequently there is more or less ataxia of the eyes.

By means of the coordinating mechanism the body is in a position to carry out each muscular function with ease and certainty and with the least possible waste of energy.

The extent and severity of the paralysis depends upon the portion of the intracranial motor path injured and the extent of the injury. As the central motor path covers a somewhat large area at its origin and local lesions of the brain tend to remain limited in extent either permanently or for some length of time, the paralysis is limited in cases of disease of the motor areas and owing to the decussation of the fibers the symptoms are exhibited by the other side of the body. The paralysis tends to be monoplegic in type, affecting either one side of the face or one limb. The paralysis is generally incomplete and only a more or less pronounced paresis is observed. If several centers are involved in the morbid processes there may be injury to motor areas on both sides, causing asymmetrical monoplegia. Further, it is to be observed that in cortical injuries the paralysis

is preceded by convulsions owing to stimulation of the cells before they are destroyed.

The further the disease is removed from the cortex the smaller the diseased area sufficient to partially or completely destroy the converging motor path. In the depth of the white matter and still more in the internal capsule and the crura cerebri somewhat more extensive lesions may cause paralysis of the opposite side of the body (hemiplegia). In cases where the crus, pons, or commencement of the medulla is injured it usually happens that the central motor path and the nucleus or basal process of some cranial nerve is involved, resulting in nuclear or infranuclear paralysis of the cranial nerve on the same side as well as partial or total paralysis of the other side of the body. In cases where the disease affects the nucleus of origin of the nerve or the nerve root there is paralysis of the part supplied by the cranial nerve alone.

Unilateral paralysis due to disease of the central motor path and involving muscles that work in concert on both sides of the body (eyes, mastication, muscles of the trunk) generally passes unnoticed, although the paralysis, as a rule, is incomplete in muscles that operate on both sides of the body independently and is, as a rule, most obvious in movements that are not automatic. The course followed by the extrapyramidal tract makes it possible that in addition to marked paresis of the opposite side there may be a certain degree of weakness in the muscles of the diseased side. Clinical observations and animal experiments adduce further proof that paralysis due to lesions of the central motor path not rarely disappear either completely or to a great extent in time.

The principal focal symptom is ataxia.

Under the term ataxia are included all obvious functional disturbances of muscles that are not due to loss of power and are not the result of paralysis. From this it follows that ataxia is due to some interruption in the sensory portion of the coordinating system, that is in the centripetal portion. Great care must be exercised in the diagnosis of ataxia in order to avoid the inclusion of an actual paralysis under the term. True ataxia is very rarely observed in animals. It far more frequently happens that ataxia and paralysis are present at the same time and it is very difficult to decide to what extent the abnormal movements are due to paralysis and to what extent due to ataxia.

It is customary to distinguish between peripheral, spinal, cerebellar, and cerebral ataxia, depending upon the seat of the lesion. When there is disease of the peripheral sensory nerves all the nerve fibers passing to the spinal cord or medulla and from thence to the cerebellum or cerebrum are blocked causing very severe motor and static ataxia. It would be unusual if the motor fibers in a mixed nerve were not also involved, in which case there would of necessity be paralysis also. In view of the fact that the sensory tracts run in different columns in the spinal cord it is quite likely that in cases of localized disease some of the other paths may remain intact. Owing to this, spinal ataxia may be less pronounced than peripheral, and according to the localization of the disease may be obvious in connection with movement or equilibration. References to spinal ataxia in veterinary literature are very frequent, but accurate knowledge shows that pure cases of the condition are very rare and that in the majority of instances it is associated with spinal paralysis. In cases of cerebral or cortical ataxia there is an absence of fine gradations of movement, in carrying out any movement the muscles contract with greater force than is necessary or the action may involve a greater or smaller number of muscles than is necessary with the result that the movement is clumsy. Cerebellar ataxia, which



from the point of view of diagnosis is far more important, is shown by the movements of the animal resembling those of an intoxicated person; staggering forwards, backwards, or sideways, and finally falling, and there may be swaying movements of the head. The animal appears to be trying to balance itself (figs. 80 and 81). During progression the feet are raised up high, in some cases abducted and in others adducted, advanced with a swinging motion and set down heavily. The animal cannot take food or water because the swaying movements of the head in all directions upset any vessel containing food. When the muscles have no equilibrating function to carry out, as for example when the animal is at rest and in many of the movements carried out when the animal is lying down, the symptoms of loss of equilibrium which before were pronounced apparently disappear entirely. If the movements are more closely observed even a minimal disturbance of co-ordination may be noticed.

Bilateral disease of the vestibular nerve may cause symptoms exactly resembling those of cerebellar ataxia. This has been observed in a fowl in which suppuration of the petrous temporal bone extending to the labyrinth resulted from bilateral purulent otitis. In this case the brain was quite free from lesions.

The convulsions which occur in some cases of disease of the motor areas of the cortex constitute the so-called cortical or Jacksonian epilepsy. In this condition there are tonic-clonic



Fig. 80. Dog affected with cerebellar ataxia.



Fig. 81. Dog affected with cerebellar ataxia.

(Illustrations made from two photographs, one taken immediately after the other.)

spasms of the whole body at long and variable intervals which may be accompanied by slight loss of sensibility over a circumscribed area. This loss of sensibility may be absent. The spasms always start in the same group of muscles and gradually spread to neighboring muscles or to the whole body. The lesion is situated in that part of the motor area containing the center controlling the group of muscles in which the spasms start. In a case of staggers we were able to satisfy ourselves that exceptionally a local symptom may become an epileptiform attack associated with coma and involving the whole body, especially when the attack always begins in the same group of muscles.

Spasms occur in the areas supplied by the cranial nerves when the lesions are near the nucleus or nerve root. For exam-



ple, in disease of the crura or the pons there is marked contraction of the pupils with absence of, or exaggerated reaction, to light, and possibly also strabismus. Spasms of the muscles of the neck are due to stimulation of the sensory nerves in the region of the medulla and generally result from meningitis. Finally, now and then a local symptom may be attributed to a reflex in cases in which a circumscribed portion of the body is involved.

Movement in circles is very rarely observed, but when this does occur it is always in the same direction. This symptom may accompany diseases of the portion of the central motor tract extending from the cortex to the medulla oblongata, the movement being either towards the diseased or the healthy side. Movement in circles associated with lateral bending of the head and neck is principally seen in diseases of the cerebellum or the middle peduncle of the cerebellum, and it may also be observed in association with unilateral blindness or deafness.

In cases of unilateral lesions of the anterior and medial portions of the thalamus produced artificially in cats and dogs Probst observed lateral bending of the head and movement in circles lasting for some minutes, in the first place away from the injured side and then for several days towards that side. Destruction of the tissues included between the posterior portion of the thalamus, the red nucleus and the anterior pair of the corpora quadrigemina caused bending of the head and movement in circles towards the injured side. Destruction of tissue in the region of the posterior pair of the corpora quadrigemina, the pons and the pyramidal decussation caused movements in the opposite direction while lesions of the posterior part of the pons caused rolling.

Hyperextension of the head and neck is observed in disease of the portion of the brain posterior to the cerebellum.

Rolling movements associated with rotation of the head about its long axis in the same direction are somewhat frequently observed in small animals. The animal, as a rule, lies on its side and then rolls over. The eyes are often involved, that towards which the rolling takes place being turned inwards and downwards and the other upwards and outwards. In many cases the former only is involved. In view of the fact that the nuclei of the cranial nerves are situated near the peduncles of the cerebellum there are often functional disturbances of these nerves (ocular motor, trochlear, trigeminus abducens, facialis).

Rolling movements are usually seen when the disease involves the peduncles of the cerebellum or the surrounding tissues. Experiments and observations have proved that such movements are not of necessity associated with disease of the middle peduncle. They occur with far greater frequency in cases of disease of the anterior part of the medulla oblongata and the hemispheres of the cerebellum. Probst's experiments have shown that unilateral lesions of the posterior part of the thalamus may sometimes cause rolling movements, and Montané saw them in inflammation of the corpus striatum. The latter observation is, however, not absolutely conclusive as the possibility of the presence of perhaps only microscopic lesions in the peduncles of the

cerebellum cannot be excluded with certainty. Clinical observations indicate that these symptoms are not always seen in diseases of the cerebellar peduncles, although there may be bending of the neck, movements in circles, and falling over to one side. Unilateral lesions of the vestibular nerve or of the labyrinth may cause rolling (Biehl, Authors' case).

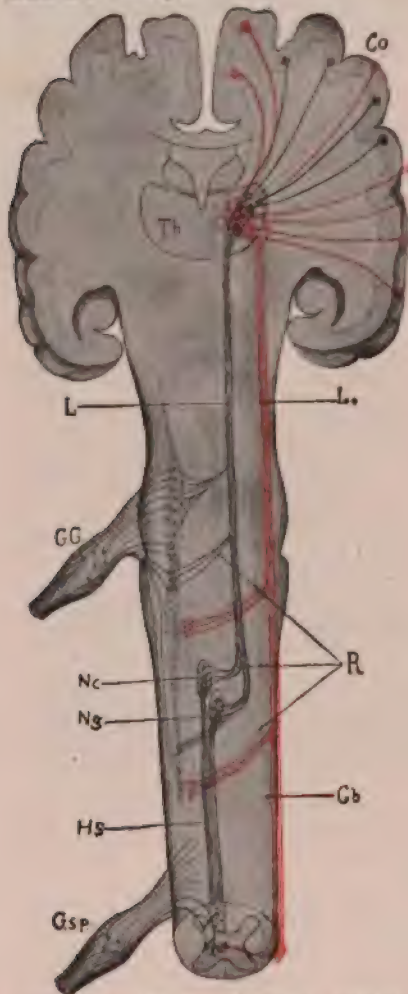


Fig. 82. Diagram of the paths of the sensory nerves (with reference to pain, pressure, temperature, taste and muscular sense). G. spl., G. G. spinal ganglion and Gasserian ganglion (the ganglia of the vagus and glossopharyngeal nerves are omitted for the sake of simplicity), with the sensory nerve paths from the skin, mucous membranes, muscles, bones and joints. The fibers passing from the spinal ganglion and entering the spinal cord through the sensory roots in part turn in the forward direction and run in the dorsal columns (black) and reach the nerve cells Nc. or Ng. (medullary nucleus of the posterior columns). From here a second path arises which crosses over at R. (raphe) and runs in L. (fillet) and is distributed to the nerve cells (black) of the optic thalamus Th. From here a third path arises (Th. Co. black) which terminates in a limited area of the cortex from which the pyramidal tract starts. A second portion of the fibers entering the cord arborise around the sensory nerve cells of the dorsal cornua. From these a tract starts (red) which crosses over to the other side and the greater part of this passes forwards in Gower's tract, a second portion in the anterior tract, Cbl. zone, and then passes through the lateral and dorsal portions of the lateral fillet. Ll. After splitting up below the posterior corpora quadrigemina this terminates in the ventral nuclei of the optic thalamus (red). From this point projection fibers (red) pass to the greater part of the cortex. The fibers of the trigeminal root arborise round the cells of the nucleus, from which a second path arises and

crosses to the opposite side, passing towards the optic thalamus in the neighborhood of the fillet. The paths and centers shown in black convey sensory impressions relating to the localized senses, while those marked in red convey the general sensory impressions (temperature, pain, pressure, etc.) (v. Monakow).

In all the natural cases so far observed the rolling is towards the diseased side but experiment has shown that it may be in the opposite direction, notably after section or simple stimulation of the vestibular nerve.

Sensibility may be impaired by lesions of the sensory tract leading to the cortex and in such cases there is, as a rule, les-

sened sensibility of the paralyzed portion of the body. In animals this partial loss of sensibility is, as a rule, difficult or impossible to recognize on account of the disturbance of consciousness which usually accompany such diseases. A symptom that is far more easily observed is the anesthesia or hyperesthesia of the portions of the skin or the mucous membranes supplied by the vagus or the trigeminus in cases where either the nucleus or the root process of either of these nerves is diseased.

In diseased conditions involving an interruption of the optic tract there are also disturbances of vision (see Fig. 83).

It has been shown by Dexler that in the horse the greater part of the fibers of the optic nerves decussate and that the proportion of the fibers from the inner portion of the retina (nasal portion) which decussate to those from the outer or temporal portion which do not cross is 5:1. Thus it happens that the optic tract and the more central portion of the nerve path of vision contain fibers coming from both eyes and the greater number of these are derived from the inner portion of the eye of the opposite side (fig. 83). The optic tract is similar to the above in the ruminants and swine, whereas in carnivora the proportion of the fibers which cross to those which do not is smaller.

Interruption of the optic nerve causes amaurosis of the eye, dilatation of the pupil and pupillary reaction is lost. Regarding the lesions induced in the chiasma, optic tract, thalamus, corpus geniculare laterale, optic radiation of Gratiolet, and center of vision by destruction of the nerve path nothing definite is known. Hemianopia has been observed by Probst in dogs resulting from lesions in the region of the thalamus produced experimentally, and by R. Balint as a result of lesions in the occipital cortex. A similar condition is seen in the human subject in cases of unilateral injury to the optic path posterior to the chiasma. Hemianopia is an insusceptibility to light in the temporal portion of the retina on the same side as the lesion, and in the nasal region on the opposite side. Total destruction of the chiasma would lead to blindness in both eyes and complete absence of pupillary reaction, but owing to the difference in the proportion of fibers which decussate in carnivora and herbivora lesions of the path posterior to the decussation determine hemianopia in the former, whereas in the latter the disturbance of one eye is far greater than that of the other. In view of the fact that in animals investigations of the power of vision cannot be so complete as in man the disturbances of vision mentioned probably pass unnoticed save in carnivora. Besnoit found total blindness on the left side in a cow affected with tuberculosis and softening due to thrombosis of the right hemisphere.

In cases where there is disease of the optic path between the chiasma and the

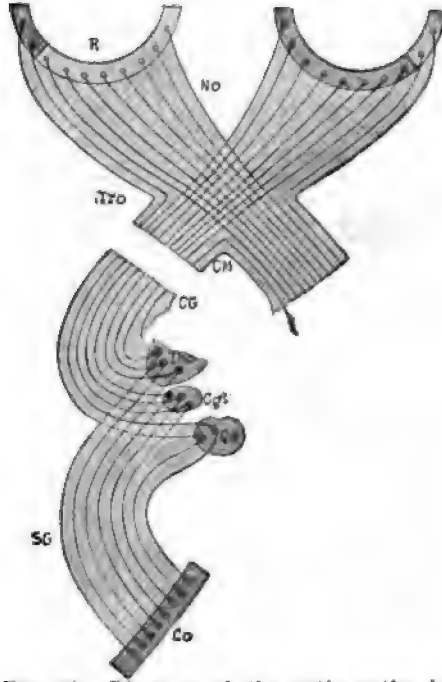


Fig. 83. Diagram of the optic paths in the horse. R. Retina of the left eye, dark portion showing the uncrossed fibers and light portion showing the crossed fibers. No. Optic nerve. Tro. Commencement of the tract behind the chiasma. CM. Meynert's commissure. CG. Gudden's commissure. Tho. Optic thalamus. Cgl. Lateral corpus geniculare. Qa. Anterior corpora quadrigemina. SG. Optic radiation of Gratiolet. Co. Occipital cortex (optic center). (Adapted from Dexler and Obersteiner.)



basal ganglia and including the latter disturbances of pupillary reaction are to be expected, although this is difficult to determine in animals because the constant movement of the eye makes it scarcely possible to use the ophthalmoscope. The diminution or absence of pupillary reaction is comparatively easier to demonstrate in animals in which the proportion of decussating fibers is somewhat large. Diseases of the cortical paths of vision cause no disturbance of pupillary reaction.

Disturbances of the senses of hearing, taste or smell sometimes occur in diseases of the brain, but they can only be discovered with difficulty, if at all, in the dog.

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## 1. Inflammation of the Pia Mater. Leptomeningitis.

(*Acute meningitis, Encephalitis, Acute hydrocephalus, Sub-acute encephalitis, Meningo-encephalitis.*)

Leptomeningitis is the term applied to inflammatory processes which commence in the pia mater, but which, on anatomical grounds, tend to spread to the superficial layers of the brain substance.

Of the meninges only the arachnoid and the pia mater tend to become the seat of primary disease. This may be due to the richness in blood of the pia mater. In view of the fact that small blood vessels penetrate the superficial layers of the brain substance from the pia mater, the diseased process is almost certain to spread to the brain and practically every case is one of meningo-encephalitis. But such cases may be included under the term meningitis in view of the fact that the process starts in the membranes, the brain being affected secondarily.

**Occurrence.** Simple acute meningitis occurs more frequently in the horse than in other animals and cases are most frequent during the hotter periods of the year. On the other hand there is no particular difference in the incidence of secondary meningitis in the different species.

According to Christian there was an increase in the number of cases of primary meningitis simultaneously with every addition of horses to the stables in the Prussian army and the number of cases increased in proportion to the increase in the number of horses. The disease reaches its maximum during the second and third quarters of the year just as does the so-called Borna disease.

**Etiology. Simple meningitis** (primary or idiopathic meningitis) is with few exceptions due to an infection, the infective material reaching the brain by way of the blood stream. The causes of the disease are only imperfectly known.

In the horse primary meningitis is not rarely a sporadic case of enzootic cerebro-spinal meningitis and is set up by the cause of that disease. This view receives strong support from the more recent investigations of Christiani. This author found diplococci agreeing with the cause of Borna disease on the one hand and with the micrococcus of cerebro-spinal meningitis of Weichselbaum in all its characters on the other, in the spinal fluid of practically all his cases of so-called acute hydrocephalus in the horse. The diplococci were never found in horses suffering from other diseases of the brain.

There is no room for doubt that for certain cases of pure meningitis in the horse and in other animals other infective materials are responsible. On the other hand, observations made on the human subject show that organisms present in the healthy body may play some part in the production of the disease, either owing to some exaltation of virulence, or owing to some decrease of resistance on the part of the body. Streit found the bacillus necrophorus in meningeal exudates of two horses.

According to Poulsen sporadic cases of simple meningitis occur in cattle at all periods of the year. Cases are more frequent in young animals than in adults and the cause is not exactly known. Poulsen further observed cases following calving which were apparently due to this, but quite distinct from parturient paralysis. This disease he named puerperal meningitis. The disease developed without exception one or two days after calving, never later, and never before calving. Christiani found diplococci comparable to the streptococcus of Borna disease in a goat affected with acute meningitis.

Misch found cocci in two cases of meningo-encephalitis in the rabbit, cultures of which set up a similar disease by intravenous inoculation into experimental animals.

According to Klee a special form of acute meningitis occurs in the pigeon (the so-called staggers [see disease of the vestibular nerve]) caused by a bacterium measuring 1 to  $1\frac{1}{2}$   $\mu$  in length.

There are certain exciting causes that play some part in the production of simple meningitis. Among these may be mentioned: bad weather (cold and wet), chills, concussion of the brain, long exposure to the sun, transport by train, intensive feeding, overexertion, etc. Up to the present no exact observations have been made to ascertain to what extent the direct rays of the sun may be responsible for meningitis. Cases are principally observed in young animals and in geldings that are housed in hot, damp stables, and fed on a rich diet; circumstances which predispose to infectious disorders. Christiani, on the other hand, was unable to show that even a long period in a stable that was damp had any predisposing effect. It is a

especially in the membranes, by a cellular infiltration. The discovery made by Dexler in a case of the so-called Borna disease associated with the investigations made in human medicine (Schultze) indicate that when the inflammation is not suppurative it may affect the deeper portions of the cortex in some cases and the summits of the gyri in others. This affects the macroscopic characters of the lesions, as in the latter case the alterations are clearly visible to the naked eye. Chemical substances which are directly injurious to the nervous tissue, and the effect of which on the blood vessels has started the inflammatory process in association with the increased intracranial pressure due to hyperemia and extravasation, cause a series of symptoms indicative of cerebral disturbance. In view of the fact that the lymph spaces of the subarachnoid are in communication with each other and with those of the spinal cord the inflammation easily spreads to the whole of the surface of the brain and generally extends to a less degree to the meninges of the cord. The roots of the cranial nerves passing through the membranes are very often involved. The stimulation of the nerve endings in the membranes due to the inflammation and the increased intracranial pressure cause pains in the head.

**Anatomical Changes.** The acute inflammation may in certain cases remain localized though, as a rule, it tends to become diffuse. The congestion of the vessels may be so slight that a definite diagnosis can only be arrived at by means of microscopic examination. In the great majority of cases there is an accumulation of more or less turbid exudate which may be either colorless or reddish in between the dura and the arachnoid and sometimes under the pia mater (hydrocephalus externus). Far more rarely the exudate is fibrinous and surrounds the dilated blood vessels, making them appear as yellowish-white streaks. In cases of diffuse meningitis all these lesions are found, involving also the pons and medulla and to a less extent the membranes of the spinal cord. The lesions may be particularly prominent in the anterior portions of the hemispheres in cases of primary meningitis in cattle (Poulsen). As a general rule pus tends to collect at the places where the inflammation started, causing the pia mater to appear as a thick membrane saturated with pus, while round about there is evidence of hyperemia and serous inflammation only (purulent meningitis). In cases of tuberculous meningitis there are, in addition to purulent and gelatinous infiltration of the meninges, grayish translucent or yellow tubercles varying in size from a poppy seed to a hemp seed along the course of the larger vessels at the base of the brain.

The cortex and the contiguous white matter appear saturated with serous liquid, moist, and swollen, and in consequence of this the convolutions appear less prominent. On the cut surface are visible vessels extending inwards from the pia



mater and petechiæ, the majority of which are caused by the escape of blood from vessels that have been cut through and which may be wiped away. More rarely there are small hemorrhages which cannot be wiped off. In exceptional cases there may be smaller or larger softened centers.

Almost without exception the venous plexuses in cases of inflammation of the pia mater appear gelatinous, while the ventricles of the brain contain a yellowish, turbid liquid which sometimes contains flocculi or fibrin or even pus (hydrocephalus internus acutus). If the inflammation is confined to the venous plexuses and the choroid plexuses (meningitis internal) the ventricle wall is sometimes softened.

In chronic inflammation of the pia the membrane appears thickened and opaque, especially along the course of the vessels, and it is closely adherent to the cortex.

**Symptoms.** **Acute meningitis** is accompanied by general symptoms of brain disturbance which gradually reach their maximum within a shorter or longer period. At the commencement disturbance of consciousness shows itself by the dullness of the animal; they take less notice of their surroundings, but stand with staring eyes and frequently take up unnatural positions. They do not respond to accustomed calls, a sudden noise startles them, but they promptly fall into a sleepy state again. Animals can only be induced to move with difficulty, and while moving are difficult to lead; their movements may be clumsy and stumbling, or in some cases the feet are lifted high and the head is held up. Dogs are particularly restless and frequently change their position and attempt to hide themselves. They make no response when called and do not recognize their owners. They are likely to snap at one's hand.

From time to time and frequently within a few hours of the onset of the disease there are symptoms of excitement and even of mania. Horses attempt to walk through or over any object that may stand in their way; they sometimes stand with their heads pressed up against a wall and move their limbs as if they were walking. In many cases they kick and bite at the manger, neigh, rear, move in circles, and finally collapse, or in some cases fall over backwards, by which time they, as a rule, have several wounds about the head and face. In cattle there is sudden restlessness, stamping of the feet, the head is held up and the appearance is wild and staring. They bellow, stagger, strike out at any object or person near them with their horns, lash their tails, and climb into the manger. Finally, they go down and general convulsions set in which may be started by a very slight external stimulus, and which recur a number of times. In cases of tuberculous meningitis, symptoms of excitement are, as a general rule, absent. Swine utter piercing squeals, root about in the straw, and try to climb the walls of the sty. Symptoms of mania occur far more rarely in the other species of animals.

In dogs such symptoms are exceptional; they wander about in an aimless fashion, howl and sometimes bite at the bars of their kennels.

The period of mania is, as a rule, of short duration, not more than a quarter or half an hour, and is followed by a period of deep depression. The animals are utterly indifferent to their surroundings, and if made to move, do so with the greatest unwillingness. They lie stretched on the floor, lifting their heads now and then and moving their legs as if walking, or they stand with eyes closed and the head supported either against the wall or in the manger. The limbs take up unnatural positions, either widely separated or even crossed, the result of which is that they collapse.

The respiration during the period of excitement appears to be accelerated, but afterwards is, as a rule, retarded, deep, and toward the end is sometimes of the Cheyne-Stokes type. The pulse may be accelerated or slower than normal, and it is full. The frequency is altered by the very slightest of influences. In the later stages it is weak and scarcely perceptible.

In carnivora and swine there is sometimes vomiting, particularly in the later stages.

At the commencement of the disease, animals, and especially dogs, appear to be in a state of hyperesthesia. With the increase of the disturbance of consciousness this decreases until strong stimuli may provoke no reaction. In cases of extensive meningitis there is often increased sensibility and warmth of the roof of the cranium.

Local symptoms are rarely observed and then, as a rule, in the later stages only. Cramp of the muscles of the eyes causes nystagmus, rolling of the eyes or strabismus. In cattle the strabismus tends to be a convergent one. The pupils are generally unequally dilated, and the reaction to light is either slow or entirely absent. These symptoms are sometimes associated with spasms of the muscles of mastication or trismus. In a proportion of cases there are contractions of the muscles of the lips, alæ of the nostrils and ears, while in some cases there are spasms of the neck. Fibrillar twitchings or muscular contractions are also seen in some cases in the muscles of one or other of the limbs. Individual muscles may become paralyzed. In this connection the squinting due to paralysis of some of the muscles of the eyes is of importance, because when associated with unequal dilatation of the pupils and rigidity it indicates some diseased process involving the base of the brain. In addition to this there may be observed paralysis of the pharynx, of the muscles of the face, tongue and levator of the upper eyelid. Very rarely there is hemiplegia.

There is often an elevation of temperature, this being one of the first symptoms. In the larger animals a rise of temperature occurring in the later stages is not infrequently connected with some septic infection or pneumonia.

There is partial or complete loss of appetite. In their moments of semi-consciousness animals will pick up food, but they chew it in a clumsy fashion and let it fall out of their mouths again, or appear to forget that they have it in their mouths. While drinking they will lower their heads into the water until their nostrils are covered. As a general rule the animals are constipated and the elimination of urine is retarded.

Careful clinical observations have shown that during the course of an attack of cerebral meningitis, there are often pronounced symptoms of spinal meningitis.

At the present time little is known regarding the symptoms of **chronic cerebral meningitis**. In view of the fact that it is the cortex that is chiefly involved disturbance of the functions of the cortex might be expected. Some cases of staggers might be due to chronic peri-encephalitis. Lecarpentier observed twisting of the head and rolling to the right in a young dog affected with ossification of the tentorium cerebelli and compression of the motor branch of the trigeminal nerve.

**Course.** The symptoms vary from case to case and make their appearance in varying sequence. Horses die, as a rule, within two or three days after the first appearance of symptoms; that is to say, before the excitement or stupor has become sufficiently pronounced to allow of a sufficiently accurate diagnosis to be made. There are cases, however, in which the onset of the mania is apparently sudden; this being quickly followed by a period of great depression and death within twelve hours. There are still other cases in which the animals appear merely exhausted for several days, the exhaustion being associated with loss of appetite, difficulty of moving, dullness, etc.; the symptoms peculiar to the disease becoming pronounced towards the end of the second or third week. There are all gradations between these two extremes and in practice the differentiation between the acute and chronic forms is not sharply marked and is of little actual value. In cattle primary meningitis generally lasts for one-half to two days, whereas tuberculous meningitis is, as a rule, subacute.

With regard to the sequence of the symptoms it may be said that the disease generally commences with dullness, followed by a period of excitement. This in turn is followed by loss of consciousness and in a proportion of cases by paralysis. These stages frequently merge into each other and there may be a complete absence of paralysis. In other cases the periods of excitement and stupor alternate at varying intervals, and not rarely there is obvious improvement in the animal. In a horse affected with leptomeningitis of the medulla oblongata Fröhner observed that the head was suddenly held in an oblique position and there was also paralysis of the muscles of deglutition with-



out any sensory disturbance. Pronounced improvement may be observed in protracted cases. The animal may improve to such an extent that it may return to work, but after a time the disease may reappear and possibly in a more severe form, death occurring shortly after. Such relapses may be due to great heat or to some external stimulus.

Complications are of frequent occurrence, the most common of which are hypostatic pneumonia or pneumonia due to foreign bodies, septicemia or pyemia.

**Diagnosis.** In well-marked cases diagnosis is not, as a rule, attended with any difficulty, especially if the history of the case affords any information as to the nature and the manner of the infection. In this connection the following points are of importance: suddenly occurring disturbances of consciousness, squinting due to disease of the nerves controlling the muscles of the eyes, contraction or unequal dilatation of the pupils, spasms of the muscles of the neck, trismus, active congestion of the papilla of the optic nerve, paralysis of the cranial nerves which is present in some cases, vomiting in the dog and pig, and painfulness of the top of the cranium. If the disease is not fully developed and only symptoms of a general nature are to be observed diagnosis presents considerable difficulty and may be impossible.

Dullness and stupor are observed in a number of infectious diseases, but in these cases the symptoms of brain disturbance are not pronounced and there is an absence of impulsive movements. In malignant catarrh the disease of the eyes (opacity of the cornea and fibrinous iritis) and the nose supplies a satisfactory explanation of the direct cause of the dullness. The differential diagnosis of rabies, suspicion of which may be roused by the attacks of mania, is based principally upon the absence of muscular spasms and the fact that rabies terminates fatally in eight or nine days at the most. Simple acute cerebral hyperemia may sometimes cause symptoms of excitement and dullness to a slight extent. Differential diagnosis in this instance is based upon the rapid and favorable course run by the disease. In the absence of a complete series of characteristic symptoms it is scarcely possible to distinguish between simple, acute meningitis and purulent meningitis. In the case of the horse acute relapses of chronic dropsy of the ventricles have to be taken into consideration. The primary meningitis occurring in cows at calving differs from parturient paralysis in that there is fever, the pupils are contracted and in some cases there are general convulsions.

Certain poisons (lead, santonin and bacterial toxins) sometimes cause meningitis which is so similar in its characters that a diagnosis can be made only by a postmortem examination. In the dog pentastomatosis and helminthiasis must be taken into consideration, and in the ox and sheep staggers. Deep-

seated inflammation in the neck sometimes causes spasms of the muscles and other symptoms, but in such cases there are no disturbances of consciousness, such as are seen in cases of meningitis.

Accurate differentiation between serous and purulent meningitis is only possible if the circumstances suggest that the infection is of a purulent nature. In cases of suppuration the symptoms are generally very severe and death takes place in a very short time. Tuberculous meningitis may be diagnosed with certainty if tubercles can be discovered on the iris or in the posterior portions of the eye. In cattle the presence of tuberculous meningitis is very probable when there are disturbances of function of the nerves leaving the base of the brain, or symptoms of a slowly progressive meningitis appear in an otherwise tuberculous animal.

**Prognosis.** Meningitis is a dangerous disease in that recovery rarely occurs save in the case of puerperal meningitis in the cow, in which instance a cure may often be effected, provided treatment be commenced in time. The longer treatment is delayed the more unfavorable the prognosis, and should symptoms persist for more than five days recovery is scarcely to be expected. About 75 per cent of cases terminate fatally and in the others, although acute symptoms disappear, some secondary disease almost always remains which greatly decreases the value of the animal. In the horse dropsy of the ventricles is possibly the commonest sequel. It may appear immediately after the disappearance of the acute symptoms, or its appearance may be delayed for weeks or even months. It sometimes occurs after repeated acute relapses. Among the sequelæ may be mentioned: amaurosis, deafness, staggers resulting from atrophy of the cortex, paralysis of individual muscles or groups of muscles.

In animals that recover there is a tendency to relapses.

**Treatment.** The first thing to do is to get the animal into a dark, quiet place where it can move (restraint only excites it), and where it cannot injure itself should it have an attack of delirium. In fine weather animals may be placed in the open in enclosures. Injuries to the head may be avoided by applying bandages. The straw should be short so that the animal may not get entangled in it. Dogs must be confined in roomy kennels so that they cannot bite people or other animals.

Herbivora should be given good hay and roots and fresh green food as far as possible, with occasional bran mash or gruel. Carnivora should have milk, soup, and boiled salt meat finely minced. Brandy and wine are useful in small animals. If the animals do not take food of their own accord they must be fed (see page 123), provided that it does not excite them. Animals will sometimes take sufficient food if it be placed in

their mouths. With the idea of supplying nourishment for the nerve cells Fambach gave horses daily injections of water or salt solution containing 0.5-1.0 gm. of lecithin which had been previously dissolved in alcohol. As a rule four or five injections sufficed. Subsequent investigations showed, however, that this had no special action. The bowels may be regulated by the administration of salts, enemata or castor oil, and cats and dogs should be given calomel (0.3-0.4 gm. or 0.05 gm.). Where there is loss of consciousness or difficulty in swallowing owing to pharyngeal paralysis, the drugs may be introduced with the food by means of an esophageal tube.

Cold water, snow, or broken ice may be usefully applied over the frontal and parietal regions. This is best done by means of rubber bags or bottles. In cases of severe and persistent stupor cold douches may be given for periods of five or ten minutes. These methods must be applied only if they can be adopted without exciting the animal.

In the very early stages bleeding may be practiced; Klemm advised the subcutaneous injection of pilocarpine (0.2-0.4 gm. for a horse) in order to reduce the blood pressure in the brain. This treatment has often been adopted, although good results have not been obtained as a rule. Alleviation or even considerable improvement has been observed in subacute cases following injections repeated for several days. Under certain circumstances the drug is prejudicial. Arecoline may be given with the same object, the dose being 0.06-0.8 gm. Potassium iodide or other iodine preparations may be given to accelerate the absorption of the exudate, large animals receiving 10-15 gm. and small animals 1-2 gm. daily.

Smart rubbing of the neck and the inner surfaces of the thighs is advised by many authors and especially in France. Intravenous injections of tincture of veratrine are of doubtful value.

Where there is great restlessness chloral hydrate given in enemata, and subcutaneous injections of morphia are indicated because the animal is likely to injure itself severely. Morphia may stop vomiting in the dog and pig.

Surgical intervention will probably prevent the extension of secondary meningitis where that is due to injuries to the cranium or to disease of neighboring organs.

During the period of convalescence the animals should be nursed with great care in order to avoid any relapse, and they should be cautiously put to work only after all disturbances of consciousness have completely disappeared for several days.

**Literature.** Argyle, *Vet. Journ.*, 1904, 162.—Augustin, *Rev. Gén.*, 1905, V, 264.—Besnoit, *Rev. Vét.*, 1906, 641.—Cadéac, *J. Vét.*, 1901, 14; 1902, 193; 1907, 588.—Christiani, *A. f. Tk.*, 1909, XXXV, 253 (Lit.).—Fröhner, *Monh.*, 1908, XIX, 129.—Hamoir, *Ann.*, 1906, 391.—Hess, *Schw. A.*, 1896, XXXVIII, 198.—Misch, *V. A.*, 1903, CLXX, 158.—Poulsen, *Maanedsskr.*, 1908, XIX, 182; *Pr. Mil. Vb.*, 1899-1908.—Rabieaux, *J. Vét.*, 1901, 729.—Siedamgrotzky, *S. B.*, 1888, 20.—Streit, *B. t. W.*, 1906, 385.—Thomassen, *Ann.*, 1893, 243.



## 2. Epizootic Cerebro-spinal Meningitis. Meningitis cerebro-spinalis enzootica.

(*Borna Disease.*)

Epizootic cerebro-spinal meningitis of the domesticated animals is an independent infectious disease characterized by inflammation of the membranes of the brain and spinal cord and the adjacent nerve tissues. In certain districts the disease is enzootic and even tends to be epizootic. In many cases there are no macroscopic lesions.

Under this term we shall here consider only those cases of meningitis which are capable of spreading epizootically, although such cases may, under certain circumstances, occur sporadically. This comprehensive definition meets both scientific and practical demands. It has been proved that in diffuse cerebral meningitis the inflammation often spreads to the membranes of the spinal cord to a varying extent, the dissemination of the infective material being favored by the communications which exist between the subarachnoidal spaces of the two membranes. It is not advisable to separate such cases from those that affect the membranes of the brain only and class them with simple cerebro-spinal meningitis. For on the one hand it is quite possible that in one case the inflammation may remain restricted to the membranes of the brain only and in other cases extend to the membranes of the spinal cord, on the other hand cerebral meningitis is as a rule secondary to some other primary diseased condition and does not spread epizootically. It is possible, however, that some forms of secondary meningitis, such as are met with in influenza and strangles, may in some cases become epizootic, but in such cases the secondary nature of the disease is obvious.

Examination of the facts regarding the pathogenesis of this disease indicates decisively that the so-called Borna disease must be ranged with enzootic cerebro-spinal meningitis.

Schneidemühl objects to the description "Borna Disease" on the grounds that it is not historically correct. The disease did not first occur in the Borna district, but had already been observed in other parts of Germany and in other countries. "Cramp of the neck" (Genickstarre) is also not so good a term to apply to the so-called Borna disease as to some other kinds of cerebro-spinal meningitis as the symptom is not infrequently absent. "Inflammation of the brain and spinal cord" cannot be considered as quite correct, for as a matter of fact when inflammatory lesions are present the affection of the brain substance is secondary. A more correct term would be "Meningo-encephalitis and Meningo-myelitis."

**Historical.** The disease of the horse observed in Württemberg in 1813, and subsequently described by Wörz as "Heat-stroke" (Hitzige Kopfkranheit) was probably an enzootic cerebro-spinal meningitis. The disease which spread through Europe in the years 1824-1828, and which was described by Franque as "Fever of the Nerves" was probably the same. Sporadic and enzootic cases of the disease were observed in several of the northern American States by Large (1847) and later by Liautard (1869). In 1878 and 1879 it attracted the attention of the veterinary surgeons in Saxony, especially in the dis-

tricts of Zwickau, Plauen, and Auerbach, and during the period 1883-1886 it became epizootic. From 1894 onwards it spread more widely and became more malignant in character, and especially in and around Borna. This accounts for the name Borna disease. In 1880, Kocurek observed an epizootic cerebro-spinal meningitis in a district in Upper Hungary, where in 1875-1876 Raisz had seen an outbreak of cerebro-spinal meningitis in the human subject. The disease is of frequent occurrence in Belgium (Mareq, "Mal d'Aizeau").

In 1896 Siedamgrotzky and Schlegel, and Johnne, simultaneously carried out extensive bacteriological and pathological investigations as to the nature of the disease. Further researches were made by Oster-tag in 1900. Wilson and Brimhall investigated the disease from clinical, bacteriological, and anatomical points of view during an outbreak in North America (1898-1903). Streit (1902), Harrison (1905), and Christiana have also made investigations. Histological investigations by Dexler (1900), and more recently by Oppenheim (1907) have furnished proof that Borna disease is an inflammatory one.

In 1867 an enzootic cerebrospinal meningitis was described by Meyer in **cattle**. This appeared to be primary in some cases, and there was never any plastic exudate. Schmidt (1888) saw cases in which there were hemorrhages of the pia mater, and the cerebrospinal fluid was gelatinous; while Utz (1896) in a similar enzootic found no pronounced lesions. The disease was observed in calves by Röder in 1896.

Cases of a contagious nature were observed in **sheep** by Stöhr, Eichbaum, and Wilke on one occasion in a district where there were cases of cerebro-spinal meningitis in man. Roloff (1868) observed it in a flock of lambs. Microscopic examination showed only perivascular cellular infiltration of the pia mater. Schmidt (1868) described an epizootic among sheep in Eastern Prussia, in which there were punctiform hemorrhages of the pia mater and cellular infiltration of the membranes of the brain. Popow (1882) and Wischnikowitsch (1889) believed that in an enzootic occurring in Russia, in which there were lesions in the lungs, the possibility was not excluded that the meningitis might be secondary. Prietsch (1896) saw an outbreak in a flock following drinking from a brook which was suspected of containing the infective material of the so-called Borna disease. In 1899 Walther described the occurrence of the disease in the district around Borna. Further observations have been made by Savigné and Leblanc regarding the disease in sheep, calves, and lambs.

In 1868 cerebro-spinal meningitis was observed by Renner in the **dog**. In one town there were 20 cases, almost exclusively confined to hounds. In these cases there was a purulent exudate between the membranes. The disease was not distemper.

Several diseases more or less resembling cerebro-spinal meningitis have been described by a number of authors, but these are apparently of a different nature. Apostolides (1880) described a very fatal disease occurring in Cairo and the surrounding district (more than 500 horses, 700 mules, and 200 asses died). This disease was, according to Nocard and Leclainche, a septicemia analogous to Mas-sauah typhus. In the cases published by Pearson, Martin and Lucet, the possibility of poison does not appear to have been excluded, and McCarthy and Ravenel have apparently observed symptoms of meningitis in certain cases of poisoning.

**Occurrence.** The disease appears to be associated with certain low-lying districts, and tends to break out either every year or at intervals of a few years. The outbreaks are variable in ex-

tent. Sometimes they are very extensive and cause severe losses and at other times there are only sporadic cases. The outbreaks tend to be more severe in years when the rainfall is heavy. It is observed, as a rule, on cultivated land only. In the majority of cases the disease occurs in affected districts only on certain farms or on certain parts of a farm, and there may be only one animal affected in a stable, while in other cases there may be a large number diseased and the death rate may be high. The severity of the disease appears to vary with the seasons. As a rule the first serious outbreaks occur in January and February. The maximum is reached in May or June and from this time they gradually decline, and in the last quarter of the year there may be either sporadic cases only or the disease may disappear entirely. This cyclical occurrence, which is particularly noticeable in affected districts in Germany, is probably dependent upon the heavy rainfall and the warmth of the earth during the second and third quarters; these factors in all probability favoring the development of the cause of the disease (Liebener). The sporadic cases which apparently occur everywhere are probably an independent type of meningitis (Christiani).

In Germany the disease is especially common in Saxony, Thüringen and Württemberg. In Saxony the following districts are chiefly affected: Borna, Grimma, Zwickau, Plauen, Auerbach, Chemnitz, Leipzig, Bautzen, Glauchau and Rochlitz. In 1896 there were 1,198 horses attacked, and in the years 1903-1908 there were 439, 590, 264, 213, 1,095, and 508. In 1903 compensation amounting to \$140,536 was paid for 439 horses, and during the period 1904-1908 the compensation for 2,903 horses amounted to \$346,496. The disease has been known in the Province of Saxony for the last thirty years, but it was in 1896 that it first became widely disseminated. The districts principally involved were Merseburg and Erfurt, Delitzsch, Eckartsberga, Querfurt, Saalkreis, Merseburg, Weissenfels, Ziegenrück, Weissensee, Langensalza. The following are the numbers of cases during the period 1897-1908: 86, 137, 499, 317, 162, 81, 109, 224, 52, 62, 492, and 127. In the years 1901-1906 the disease was reported in Schwarzburg-Rudolstadt, and isolated cases occurred in Saxe-Coburg-Gotha and Magdeburg.

The disease was observed in Hungary in 1880-1882 and again in 1897 and 1898 in horses in the district of Zemplén. About 200 animals were affected. Fresh outbreaks have been reported since that time.

Two great outbreaks have been reported in Belgium.

Cases have occurred in horses in Great Britain and Russia, more than 200 horses dying in the Don Province in 1902.

In North America, New York, Pennsylvania, New Jersey, Minnesota, Ontario, and Illinois appear to be the most seriously affected. About 4,000 horses and mules were destroyed in the valleys of the Brazos and Colorado rivers.

**Etiology.** Epizootic cerebro-spinal meningitis is due to an infection.

In the subarachnoidal fluid of a horse affected with the so-called Borna disease, Siedamgrotzky & Schlegel found a micrococcus and more rarely a diplococcus measuring about 0.6  $\mu$ . On the surface of gelatin this organism formed dirty white, sharply circumscribed colonies about the size of linseed. These colonies showed a characteristic dense-looking point at the center. In broth there was diffuse turbidity and a large amount of flocculent deposit. Intravenous inoculation with cultures into horses in one case produced no effect; in a second, there were



symptoms of a slight brain disturbance, and the micrococci were demonstrated in the cerebro-spinal fluid; while in a third, the injection was followed by symptoms of sleepy staggers. Subdural inoculation into horses caused severe meningo-encephalitis, the cocci being demonstrable in pure culture in the exudate.

Johne found diplococci in the cerebro-spinal fluid, and in one case in the blood of diseased horses. The cocci measured 0.4 to 0.8  $\mu$ ; and some were free and others included within cells. After staining with Ziehl's stain and washing with dilute acetic acid, the organisms showed special shapes resembling coffee beans or wheat grains. They were not constantly Gram-fast.

The cultural characters resembled those of the Siedamgrotzky-Schlegel organism and short chains were formed. Growth was particularly luxuriant in the condensation water of agar when the cultures were incubated. This organism was called by Johne, the *Diplococcus intercellularis equi*, on account of its morphological and biological resemblance to the causal organism of contagious cerebro-spinal meningitis in man. Typical symptoms of Borna disease followed the subdural inoculation of the organism into two goats and three horses. All the horses recovered while the goats died. At the postmortem of one of the goats a purulent and fibrinous spinal meningitis was discovered. Similar diplococci were found by Mareq in diseased horses in Belgium.

Organisms morphologically resembling the diplococcus of Johne were found by Ostertag in cases of Borna disease. The organisms occurred only in very small numbers, and were very rarely included within cells. They were as a rule present in the subdural and ventricular fluid of the brain and occasionally in the blood, liver and urine, but only very exceptionally in the substance of the brain. On artificial media short chains were formed; division was in two planes; the organism was non-motile, and Gram-fast. Growth on artificial media was scanty, but it was particularly abundant in the condensation water of agar. The organism grew equally well on acid and alkaline media. In broth there was uniform turbidity, contrary to what is the case with the streptococcus pyogenes. Gelatin was not liquefied. Multiplication of the cocci occurred in water containing ammonia or nitrous acid. The optimum temperature for growth was that of the body. Resistance offered by the organism was slight, desiccation causing rapid destruction. The organism soon died in pure water, while they remained alive for 4 months in moist substances. Multiplication occurred in water containing drainage from stables.

The microorganism designated by Ostertag as Borna-Streptococcus was pathogenic for the horse, but not for the laboratory animals. Subdural inoculation into horses was promptly followed by the usual symptoms. According to Profé, repeated intravenous inoculations at short intervals caused a disease resembling Borna disease, while a single inoculation or a number spread over a long time caused a condition that was not at all typical, and quite transitory. Goats and sheep are susceptible to subdural inoculation, but not to so great an extent as the horse. Cattle and pigs are refractory. The introduction of the organism under the skin, into the nose, eye, ear or alimentary canal is not followed by any symptoms in the horse.

Streit isolated an organism closely resembling, and possibly identical with the streptococcus of Borna disease from a case of epizootic cerebro-spinal meningitis in a horse in Ontario, as did also Grimm in southern Germany, while Harrison observed a quite different organ-



ism. Christiani found an exactly similar organism in primary sporadic meningitis in the horse, and in a goat (see page 625).

In an outbreak in Minnesota, Wilson & Brimhall found an organism corresponding with the *Micrococcus meningitidis cerebrospinalis* of Weichselbaum in the central nervous system of a cow. In other outbreaks in cattle, horses, sheep and pigs only Fränkel's diplococcus pneumoniae could be found. The latter organism was also found at the same time in men dead of cerebro-spinal meningitis. Horses inoculated subcutaneously, intravenously, subdurally, in the brain and lumbar portion of the spinal cord, all died showing typical symptoms of cerebrospinal meningitis; and diplococci were found in pure culture in the central nervous system of the experimental animals. A similar organism was found by Trambusti in the meningeal exudate of a sheep, by Manfred d'Ercole in two calves, and Zangheri in a horse.

Bacteriological investigations have, therefore, not as yet given any perfectly satisfactory results, although it is probable that Siedamgrotzky & Schlegel, Johne, Ostertag, Streit, Grimm, Christiani, Marcq, and possibly also Wilson & Brimhall, were dealing with the same organism which had in some way varied its characters somewhat. Further investigations are necessary to decide whether the cause of the so-called Borna disease is always present in cases of cerebro-spinal meningitis in the horse, and whether it plays any part in the production of the disease in other species, at least in a proportion of cases. The observations of Prietsch, Walther, Pröger and Wilson & Brimhall appear to indicate that this is the case. A solution is also required to the question as to what relationship exists between the organism described by Johne and others to the diplococcus intracellularis of the human subject. According to Johne the two may be distinguished by the fact that the organism which occurs in the horse may be present in the central nervous system without causing lesions but simply an intoxication. According to Ostertag there is no connection between the two organisms. Christiani, on the other hand, was unable to find any differences between the streptococcus found by him and the diplococcus intracellularis of Weichselbaum.

There is the possibility that epizootic cerebro-spinal meningitis in the lower animals is not an etiological entity, and as the diplococcus intracellularis and the *d. pneumoniae* occur in man, each may be responsible for epidemics of the disease in the human family.

**Natural Infection.** Under natural circumstances infection takes place through the medium of infected water or food. The fact that at the postmortem there is usually found catarrhal pharyngitis and gastro-enteritis suggests the possibility that, in spite of the negative results so far obtained experimentally by feeding, the infective material enters by the alimentary canal and in some unknown way gains access to the blood stream and thus is enabled to exercise its toxic effect.



Water contaminated with nitrogenous materials and all food coming into contact with it are equally dangerous in this connection. Ostertag & Profé have been able to isolate the *Borna streptococcus* from water containing ammonia and nitrous acid obtained from the drains of infected stables. According to Liebenner the organism may exist in the ground and its dissemination depends largely upon the nature of the flooring. Where the subsoil is loamy the infective material may be washed down by heavy rains and finally be carried into streams.

It is not certainly known how the infective material reached the ground or water, but, although its presence in urine is the exception, yet it is possible that it is by means of the urine of diseased animals. It is probable that under suitable conditions the organism can maintain a saprophytic existence, and that under certain circumstances that are not as yet known it becomes pathogenic.

No observations have been made in veterinary medicine as to whether the organism may be present in the upper air passages of otherwise healthy animals and under certain conditions develop pathogenic characters.

The disease is not transmitted directly from animal to animal. Some observations appear to suggest at first sight that this does occur, but it is far more probable in such cases that the infection is carried by food or water which has at some time been in contact with diseased or infected animals.

**Susceptibility.** Naturally the horse is the most susceptible animal, followed in order by the sheep, ox and goat, while the pig and dog are only very exceptionally infected. In certain outbreaks several species of animals have been affected at the same time.

Breed and sex exercise no influence on the susceptibility, while intensive feeding, especially with clover hay, appears to increase the susceptibility. Young animals appear to be more susceptible than old ones, but this is not always the case.

Recovery from one attack leaves the animal with no immunity.

**Pathogenesis.** When the causal organisms reach the membranes or the neighboring part of the cortex toxic materials, either secreted by them or set free by their disintegration, injure the vessel walls and the adjacent tissues. As a result there is set up an inflammatory process associated with cellular infiltration and sometimes hemorrhage. This holds good both for the so-called Borna disease and for other forms of cerebro-spinal meningitis. In certain cases there is only the escape of a liquid that is poor in albumen and the formation of small, scattered centers of cellular infiltration, and even these may in some cases be absent. The final result appears to be influenced not only by the virulence of the infective material, but also by the species of animal affected.



Borna disease was recognized by Siedamgrotzky as a serous leptomeningitis and by Kitt as an inflammatory process. Dexler was able to demonstrate evident signs of inflammation, namely, centers of small-celled infiltration in the membranes of the brain and cord, the adjacent layers of the nervous tissue and in the venous plexuses (see Figs. 84 and 85). The last named author considered Borna disease to be a meningo-encephalitis and myelitis. He admits that there is bacterial intoxication as in the case of other inflammatory diseases of the brain, such as rabies. In some cases the toxic effects and in others the inflammatory processes gain the upper hand, depending upon the intensity of the infection. Dexler's view was further supported by the results of the histological investigations of Wilson and Brimhall. These authors pointed out at the same time the discrepancy between the lesions and the disturbance of health, a point already remarked by Dexler. More recently Oppenheim has proved the presence of diffuse meningitis involving also the superficial layers of the nervous tissue in a typical case of Borna disease.

How difficult the discovery of the inflammatory lesions is, is shown by Dexler's case mentioned above. In this case preliminary microscopic examinations of the various parts of the central nervous system proved absolutely negative, and the lesions were only found by systematic examination of a number of pieces of the brain.

According to Schmorl and Johné, Borna disease is a pure intoxication of the central nervous system which leads to dropsy of the meninges and ventricles, owing to injury to the endothelial lining of the vessels caused by venous stagnation. In view of the fact that both in natural and in experimental cases the streptococcus of Borna disease occurs but sparingly in the cerebro-spinal fluid, Ostertag believes that the organism is destroyed in the blood of the animal, and that the toxin thus liberated is able to exert its effects upon the nervous tissues.

Apart from the toxic effects the nervous tissues are injured more or less severely by the products of the inflammation. The escape of liquid raises the intracranial pressure and pronounced disturbances of function are caused by the cellular infiltrations.

**Anatomical Changes.** In susceptible animals the only lesions that are found are, as a rule, dilatation of the veins in the pia mater of the brain and cord and an increased quantity of clear yellowish liquid in the sub-arachnoid space and the ventricles. Endothelial cells are sparingly present in the liquid and a few red and white corpuscles. In five cases investigated by Johné the percentage of albumen in the liquid varied from 0.035-0.17 per cent.

Occasionally there is localized gelatinous infiltration around the vessels. The brain substance appears more or less moist.



In one case Johne found scattered small reddish-yellow centers in the cortex and corpus striatum. The roots of the nerves appear to be uninjured.

Histological investigations carried out by Johne and Schnorl, and by Ostertag yielded negative results; Dexler in one case, and Wilson and Brimhall in all the cases investigated found a small-celled infiltration in the meninges and brain. Dexler found this especially at places where the arachnoid bridges over fissures in the brain and also in the immediate neighborhood of blood vessels in the peripheral parts of the brain and cord (see figs. 84 and 85). Oppenheim found a diffuse thickening of the pia mater due to an increase in the amount of connective tissue and also round-celled infiltrations in the pia mater and in the superficial layers of the cortex. Very occasionally there were cellular infiltrations around the blood vessels in the deeper tissues. At places there were small hemorrhages.

Peculiar "nuclear inclusions" were demonstrated by Joest and Degen in the cells of the ganglia. Applying a modification of Mann's stain for the demonstration of Negri bodies, the large ganglion cells of Ammon's horn showed small bodies staining intensely with eosin and containing an unstained part. These bodies were as a rule rounded but sometimes oval and occasionally divided into two halves. The nature of these bodies which are apparently characteristic of epizootic cerebro-spinal meningitis of the horse is not known.



Fig. 84. Disseminated infiltration with leucocytes of the ventral column of the first cervical segment of the cord in a case of Borna disease. Enlarged 30 times, (Dexler).

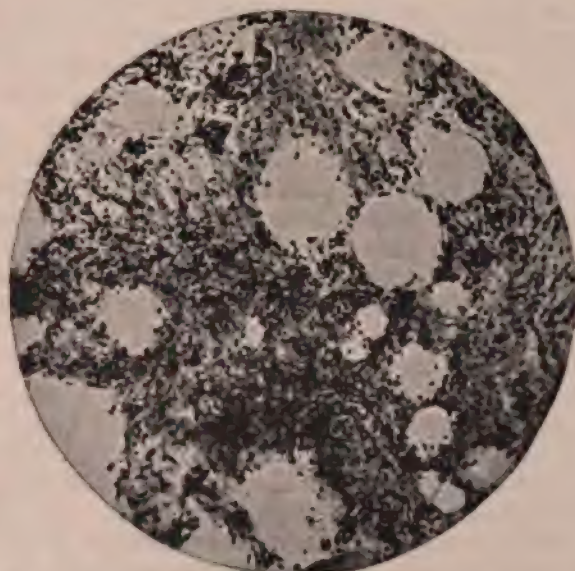


Fig. 85. Round-celled infiltration and lacuna-formation in the pia mater of the cerebrum in Borna disease (Dexler).

In the other species the lesions are similar to those found in the horse, or as is sometimes the case in that species, there may be a yellowish-white fibrinous or purulent exudate along the course of the vessels of the membranes covering the base



of the brain and medulla. The cerebrospinal fluid in such cases appears turbid and contains flocculi or fibrin or pus.

**Symptoms.** Little is definitely known regarding the period of incubation. Noack observed symptoms in a horse from a healthy district nine days after its introduction into an infected district. In some cases there are digestive disturbances, loss of appetite, frequent gaping, jaundice (very characteristic according to some authors). In the horse there may be symptoms of colic, catarrh of the pharynx and respiratory tract; these being accompanied or followed by gradually increasing depression and dullness which continue to the end. During this period the animals may blunder into objects in front of them and fall down. From time to time there may be attacks of vertigo which may cause immediate collapse.

Symptoms of excitement are observed and these are principally due to external stimuli. This may manifest itself either as fear, pronounced restlessness, or even in exceptional cases in attacks of mania. For the rest the symptoms of excitement resemble those of meningitis (see page 600), although they may not appear until the end.

Within a short time, and in some cases quite early, spasms occur in certain groups of muscles of the head. There may be squinting, unequal dilatation of the pupils (rare), spasmodic elevation of the alæ of the nostrils and lips. Very frequently there is grinding of the teeth, intermittent trismus, etc. To these is added difficulty in swallowing from the very outset and it may be the most prominent symptom. Animals retain their appetite and take in food and chew it, but are unable to swallow the food or saliva that collects in the mouth, and as result there is a flow of saliva from the mouth. In some cases the food collects in the pharynx and causes severe pharyngitis. There are often spasms of the muscles of the neck, the head being drawn back by the tense muscles, and the animal rests with the breast, the lower border of the neck and the chin against the wall. In the horse there are sometimes spasmodic contractions of the muscles drawing the head back. In some cases the contractions are more powerful on one side with the result that the neck is drawn to that side. It is useless to try to overcome the muscular cramp and get the neck into the proper position, such attempts only increasing the spasms. Not uncommonly there is only a more or less obvious stiffness of the muscles, which may be discovered when attempts are made to move the head passively, and frequently the spasms are quite absent. With the extension of the inflammation along the cord the muscles of the back are similarly affected. In many cases brief contraction of the muscles of the back and other parts of the body are observed at short intervals (Schmidt). In many cases the spasms principally affect the extremities and the vertebral column may be curved throughout its length (opisthotonus).



Sensibility may be increased at the outset but later, and not rarely from the beginning, gradually decreases in proportion to the dullness. In certain parts of the body, neck, along the vertebral column, etc., there may be pronounced hyperesthesia up to the last. Animals show this increased sensitiveness by rubbing or biting the particular parts. Reflex irritability is increased at the commencement and this may persist throughout. Slight external stimuli are sufficient to cause spasms of certain muscles and even general cramp. In entire animals and even in castrated animals there may be frequent or persistent erections, and in mares the corresponding symptoms are seen.

In some cases there is paralysis. Paralysis of the muscles of the face causes distortion of the features. The tongue hangs out of the mouth and swallowing is impossible. In many cases there is weakness of the hind quarters.

The temperature is variable, but is, as a rule, somewhat elevated, and in the horse oscillates between  $39^{\circ}$  and  $39.5^{\circ}$  C. Extraordinarily high temperatures are only rarely encountered. In one case observed by Mergel and Knabe the temperature was  $41^{\circ}$  C., and in one seen by Ekkert  $41.6^{\circ}$  C. It commonly happens that there is a rise of temperature at the outset only, there being a fall to the normal afterwards. Sometimes the temperature rises to a marked extent at long intervals. Persistent high temperature indicates a complication of some sort.

The rate and rhythm of the heart's action are variable, comparatively slight external stimuli causing marked acceleration. Marked and persistent acceleration of the pulse indicates either a complication or paralysis of the vagus. Respiration may be either more rapid or slower than in normal animals.

Appetite is, as a rule, poor from the outset. Prehension of food is rendered difficult, owing to cramp of the lips and muscles of mastication and paralysis of the pharynx. Diseased horses are unable to use their lips in the prehension of food and they bite right into the food, mastication being frequently interrupted. There is constipation and the abdomen appears drawn in.

In the later stages the gait is very uncertain, and in some cases movement is impossible, the animal lying unconscious on the ground. Respiration is shallow and rattling and sometimes of the Cheyne-Stokes type, the limbs are extended and are sometimes moved in the horizontal direction (so-called swimming movements). There may or may not be symptoms of paralysis or cramp at the time of death. The immediate cause of death may also be due either to general sepsis following bed sores, pneumonia due to foreign bodies or fracture of the skull.

According to Kühn, the symptoms of Borna disease are very variable. In some cases there is hemiplegia, in others rapidly progressive paralysis of the lips, tongue and pharynx; and in still others, a striking change in the disposition of the animal, previously quiet horses becoming vicious (as seen in a case by Kalkhoff). Now and then, horses will



not go forwards, or they flex the joints of their limbs that the body almost comes into contact with the ground.

In some cases there is a vesicular eczema (Fambach, Knabe).

**Course.** The symptoms described do not always follow in the order given, nor are they all present in every case. They vary considerably in severity. As a rule the inflammation starts at the base of the brain and extends to the medulla and spinal cord, but sometimes the order is reversed. In six cases seen by Noack the disease commenced with paralysis of the quarters. The inflammatory processes may remain more or less localized up to the time of death, spinal symptoms being consequently absent. In other cases these may be the more prominent.

The duration and termination of the disease are also variable. In the mildest cases there is simply dullness for five to eight days, followed by complete disappearance of the symptoms. On the other hand, there may be severe spasms or even paralysis on the first or second day, death following after a short interval. On an average the disease lasts from one to two weeks, symptoms gradually coming to a crisis, or they may abate for a time and finally disappear. In many cases slight symptoms persist for some length of time. In certain cases there is a temporary improvement, followed by a relapse, especially when the animal is put back to work.

In well-marked cases the disease tends to be fatal, although the mortality varies in different outbreaks within wide limits. Kocourek observed recovery in 50 per cent of cases, while in the case of Borna disease and even in milder outbreaks at the most 20 to 25 per cent of the animals remain alive, and sometimes the majority or even the whole of the diseased animals may die. Recovery is, as a rule, incomplete, various sequels being observed, dulness, amblyopia, amaurosis, lumbar weakness, epileptiform seizures. These may disappear in the course of months.

**Diagnosis.** Diagnosis is based upon the epizootic nature of the disease and the simultaneous appearance of symptoms due to disease of the membranes of the brain and cord. In this connection the spasms of the neck, and the presence of symptoms of disease of the bulb and spinal cord, are of the utmost importance. In infected districts the diagnosis may with great certainty be based upon a far less complete series of symptoms. The symptoms described differentiate the disease from cerebral meningitis, including hydrocephalus internus, tubercular meningitis, staggers (cœnurosis), rabies, vertigo, epilepsy, poisoning, etc. In cases of tetanus there is stiffness of the neck and other portions of the body, but there are no clonic spasms and consciousness persists to the end.

**Treatment.** Treatment is in general principle like that for cerebral meningitis (see page 604). Cold applications, such as icebags, are the best local treatment. The spasms may be mod-



ified by the administration of anti-spasmodics, which may be given either subcutaneously or per rectum. Schmidt records the recovery of four horses following subcutaneous injections of dialysed pyocyanase. In Germany satisfactory results have been obtained in mild cases by long-continued administrations of small doses of calomel (2 gm. daily), or subcutaneous injections of turpentine. Subcutaneous injections of emulsions of healthy brain tissue are without effect. Lecithin, advised by Fambach, has been proved to be useless. Fambach and Zech, among others, have not been able to convince themselves of the value of the sublimate-atoxyl treatment advised by Dorn. Statistics show that none of these or other treatments have much effect upon the mortality of the disease. At the present moment there appears to be no prospect of treating epizootic cerebro-spinal meningitis successfully.

**Prophylaxis.** In order to hinder the spread of the disease food, water, and pasture, should be changed, and in all cases healthy animals should be isolated from the diseased, and if possible removed to a different stable altogether. Special care must be taken that the drinking water contains no nitrogenous materials, and particularly the drainage from the stables must not be allowed to contaminate the water. According to Kühn green food, roots or raw potatoes should be given to horses in affected districts and horses used in agriculture should have their feet thoroughly washed in 10 per cent creolin every night.

**Veterinary Police.** From this point of view the inclusion of epizootic cerebro-spinal meningitis among the notifiable diseases appears to be advisable. Regulations should be made with reference to improvement of local conditions, disinfection, isolation of diseased animals, and careful disposal of carcasses, but it does not appear to be necessary to limit movements of healthy animals on infected premises.

The disease has more recently been scheduled in the Kingdom of Saxony and in the Prussian Province of Saxony.

**Literature.** Christiani, A. f. Tk., 1909, XXXV, 253 (Lit.).—Dexler, Z. f. Tm., 1900, IV, 110.—Eichbaum, Stöhr, Wilke, Pr. Mt., 1865-66, 135.—Francis, Am. Vet. Rev., 1905, 148.—Görägew, Journ. f. allg. Vet. Wiss., 1906, 242.—Grimm, Unters. üb. bei d. sog. Kopfkrrh. d. Pferde gefund. Bakt. Diss. Giessen., 1907 (Lit.).—Joest & Degen, Z. f. Infkr., 1909, VI, 348.—Johns, D. Z. f. Tm., 1896, XXII, 371; S. B., 1896, 57.—Kalkoff, Z. f. Vk., 1908, 247.—Klett, D. t. W., 1898, 329 (Lit.).—Kocourek, D. Z. f. Tm., 1891, XVII, 133.—Kühn, B. t. W., 1908, 173.—Liautard, Rec., 1869, 361.—Marcq, Ann., 1909, 11.—Martin, Am. Vet. Rev., 1898, 829.—Noack, S. B., 1908, 41.—Oppenheim, Z. f. Infkrh., 1907, II, 148.—Ostertag, *ibid.*, 1907, II, 152; B. t. W., 1900, 433.—Pröger, S. B., 1896, 105.—Renner, Mag., 1868, 451.—Rolooff, Pr. Mt., 1868-69, 147.—Röder, S. B., 1896, 140.—Russinow, Journ. f. allg. Vet. Wiss., 1906, 242.—Schmidt, Mag., 1870, 186; A. f. Tk., 1887, XIII, 459; Monh., 1909, XX, 435.—Siedamgrotzky & Schlegel, A. f. Tk., 1896, XXII, 287.—Streit, B. t. W., 1903, 577.—Utz, D. t. W., 1896, 259.—Walther, S. B., 1899, 80.—Zangheri, Clin. Vet., 1904, 217.



### 3. Cerebral Hyperemia.

A number of diseased conditions have been ascribed, especially by old authors, to hyperemia of the brain, which were due either to diseases of the brain that were not recognizable to the naked eye, or to disease of other organs. Marked congestion of the vessels of the brain and the membranes were held to be sufficient to explain the symptoms. It must be remembered, however, that such congestion may be due to the head being in a position lower than the body either at the time of death or after, and experiments by John, Kitt and Dexler have shown that there may be pronounced congestion of these vessels in healthy animals or animals that have died from other diseases. The older observations cannot therefore be taken as accurate, and it remains undecided to what extent these postmortem alterations were taken into account by the old authors. In the vast majority of cases, the diagnosis was based simply upon clinical symptoms or upon references to the subject in the older literature. Examination of the causes mentioned, shows that the nervous symptoms would be of quite a different kind, and that in many cases not only would there be no hyperemia, but an actual anemia.

It cannot be doubted that variations occur in the amount of arterial blood in the brain; but, as rightly pointed out by Dexler, and as observed in the human subject, these variations are manifested more by subjective than objective symptoms. The recognition and correct interpretation of these symptoms is associated with great difficulty. It is certainly rare to find deep-seated disease of the brain unassociated with active hyperemia, which is recognizable clinically, and at the post-mortem.

**Etiology.** The following are some of the influences which may determine an increased flow of blood to the arteries of the brain: increased cardiac activity, loss of tone of the cerebral arteries, overexertion, rough handling during the process of breaking-in, severe psychical disturbances during transport by train or boat, rutting, fear, hypertrophy of the heart, acute poisoning with alcohol and other narcotics, direct rays of the sun falling on the head, too great heat, etc. A kind of collateral hyperemia is set up when large areas are cut off from a supply of arterial blood as in thrombosis of the posterior aorta or other large vessel, and when the abdominal vessels are compressed owing to tympanites. Acute inflammatory processes of the brain and membranes are always associated with hyperemia, but such instances are more conveniently considered in connection with the causal condition.

Passive congestion may be caused by compression of the jugular veins by the splenius muscle, tumors, enlarged thyroids, and any sort of inflammatory swelling. It may also be set up by a weak heart, chronic lung disease, or compression of the lungs owing to gastric or intestinal tympanites.

**Anatomical Changes.** In case of acute hyperemia of the brain and membranes the brain substance is reddish or yellow

in color, the venous plexuses of the pia are deep red and there may be echymoses. The lesion described may often be missed in spite of there having been active hyperemia during life, because it tends to disappear for the most part after death (Kitt, Dexler).

In passive hyperemia there may be observed a pronounced congestion even in the small vessels and the tortuous course of the veins may be traced on the surface of the brain, between the convolutions, and even into the cortex. Small hemorrhages may be found along the course of the vessels, an increase in the otherwise clear cerebrospinal fluid and a saturation of the tissues with serum.

**Symptoms.** In cases of **active hyperemia** the period of excitement may be followed by one of depression with repeated recurrences of excitement. In slight cases the animals are restless and excited, the eyes are bright, the pupils dilated, and the pulse and respiration accelerated. The cranium feels abnormally hot and the mucous membranes of the head are injected. There appears to be some disturbance of consciousness as is indicated by awkwardness of movement, alarm, and loss of appetite.

**Passive hyperemia** is generally characterized by depression, cyanosis of the mucous membranes, difficulty of respiration, and a small rapid pulse. It must be remembered that the brain possesses great adaptability to slowly progressive circulatory disturbances, and consequently symptoms are usually absent in cases of severe but long-standing congestion.

**Course.** Simple arterial hyperemia generally passes off within a few hours. In favorable cases the condition returns to the normal either suddenly or gradually, and within a few hours all symptoms may have disappeared. Venous hyperemia is a temporary condition in cases in which the cause of the congestion can be removed. In other cases it is persistent, increasing from time to time, and may lead to the production of great depression.

**Diagnosis.** In slight cases the normal condition of the animal, as regards its cerebral functions, its age and strength, must be considered. The periodic appearance of sexual impulse must be taken into consideration, for this sometimes causes similar changes in animals. The possibility must not be lost sight of that the symptoms of hyperemia mark the onset of some acute infectious disease, or that some organic disease of the brain, stomach or intestine may be behind it. Diagnosis and prognosis in the early stages must be made, taking into consideration the possible development of further symptoms. As a general rule organic diseases of the brain may be excluded if one or two days pass without further development. In the horse exacerbations



of chronic dropsy of the ventricles or encephalitis may be easily mistaken for hyperemia of the brain.

**Treatment.** In slight cases it is sufficient to bring the animal into a large airy box where it can be quiet, and supply it with easily digested food and fresh water.

In severe cases of active hyperemia, prompt venesection in the early stages may give very good results, cold compresses applied to the head, and irrigations with cold water may bring about the disappearance of the hyperemia. For internal treatment purgatives are indicated, but overdosing with drastic drugs may do harm.

Efforts must be made to get rid of the cause of the hyperemia if possible, and animals that are liable to be affected for any reason must be guarded against excitement.

**Literature.** Dexler, *Nervenkrankheiten d. Pferdes*, 1899, 188.

#### 4. Sunstroke and Heatstroke.

**Etiology.** Severe nervous disturbances not rarely occur in animals that are exposed to the direct rays of the sun during hot weather while at work, or driven in large herds, or transported in open cars. But similar cases also occur without the animals being exposed to the sun, the symptoms being due simply to the heat. Cases come under observation especially in the army during drill, maneuvers, or war, in droves of pigs, sheep and cattle, and during transport by rail. The occurrence of such cases is comparatively rare in the temperate zones.

The disease named by Bongartz "Summer heaves" (*Sommerdämpfigkeit*) belongs to this class of disease. This affects horses suddenly that are working in the open. A proportion of the disease termed "fatigue diseases" (*Ermüdungskrankheiten*) also comes under this heading (Bartke, *Schimmelpfennig*).

**Pathogenesis.** The direct rays of the sun falling on the cranium may possibly cause dilatation of the intracranial vessels and thus cause hyperemia, but the severe disturbances which are sometimes fatal within a short time can hardly be caused solely in this way. The possibility must be considered whether the sun's rays, the chemical and not the heat rays, cannot be the cause of the inflammatory processes. Such a possibility is difficult to conceive in spite of the fact that Amato believes that the nerve cells may be injured by ultra-violet rays. A more probable view is that the direct rays beating upon the cranium cause excessive heating of the central nervous system, thus setting up paralysis of the vaso-motor and respiratory centers. As a result of this, there is a drop in the arterial blood



pressure and respiratory disturbance. If the loss of heat be diminished owing to a high external temperature or close crowding of animals and the body heat be increased by muscular exercise, the temperature of the animal body may be raised so high ( $40^{\circ}$  to  $45^{\circ}$  C) that nerve paralysis may be produced without the direct effect of the sun at all. It is quite easily understood that a general rise of body temperature is scarcely ever absent when an animal is exposed to the direct rays of the sun. A sharp distinction between sunstroke and heatstroke is upon these grounds both impracticable and unnecessary.

Experimental evidence has recently been furnished by Marinesco that nervous symptoms may be caused both by the direct rays of the sun, and also by an increase in the body temperature in animals kept in very hot but quite dark boxes.

Christiani has demonstrated the diplococcus of sporadic meningitis in the cerebro-spinal fluid of horses that have died from heatstroke. But in these cases there may have been primary meningitis owing to the unfavorable effect of the heat.

**Symptoms.** In the earliest stages there is a rapidly progressive dullness and depression, the gait is uncertain and stumbling, sweating is observed, and the expression of the face is anxious. There is palpitation of the heart, the pulse is accelerated and weak, and respiration difficult. The mucous membranes are in the early stages injected, but afterwards become pale and even bluish. The temperature may be as high as  $45^{\circ}$  C. or even higher. In rare cases, the animals show symptoms of excitement which may amount to mania. Towards the end, there is violent trembling, the animals falling to the ground and dying in convulsions, if prompt assistance is not forthcoming. In some cases death is sudden without any remarkable disturbance of health having been observed, in other cases animals die in from one to three days.

In the so-called summer heaves ("Sommerdämpfigkeit") Bongartz observed sudden difficulty of respiration, causing heaving movements of the entire body. While going uphill or drawing a heavy load, animals often fell, and sometimes dropped dead. If brought into warm stables respiration remained difficult for some hours, and there was marked dullness. During cooler or wet weather, there was a rapid improvement in their condition. In cases where the attacks occurred repeatedly, the animals became emaciated.

**Diagnosis.** In making a diagnosis the following conditions must be carefully excluded, acute infectious diseases, and especially septicemic conditions (anthrax, swine erysipelas) encephalitis, relapses in cases of chronic hydrocephalus, epilepsy, congestion or edema of the lungs. Special importance attaches to the last two since they are likely to be set up by the same external influences.

**Treatment.** If possible animals should be placed in a shady, cool spot or in water, and cold water should be poured

over the upper parts of their bodies, and cold applications placed over the cranium. If necessary, drugs having a vasoconstrictor action may be used, oil of camphor (20 to 30 gm. subcutaneously for large animals and 1 to 5 gm. for small), caffein, (4 to 8 gm. or 0.1 to 0.5 gm. subcutaneously), small doses of ether or alcohol. If there are signs of edema of the lungs, venesection may be practiced. According to the observations of D'Anchald, the shelters found in many large towns, the object of which is to prevent the occurrence of the disease, are injurious rather than useful.

**Literature.** Bartke, D. t. W., 1898, 101.—Bongartz, B. t. W., 1889, 259.—D'Anchald, Bull., 1907, 607.—Marinesco, Compt. R., 1906, 853.

### 5. Cerebral Anemia. *Anæmia cerebri*.

**Etiology.** Acute cerebral anemia follows large losses of blood, and may be caused by excessive quantities of blood passing to other organs (too sudden escape of exudates or transudates from the large body cavities, or of gas from the alimentary canal, too rapid parturition). Anemia of the brain of varying severity may result from cardiac weakness and general dilatation of the vessels, in severe infectious diseases, or various kinds of poisoning. It is only very occasionally that cerebral anemia in animals is due to constriction of the vessels of the brain owing to some psychic influence or severe irritation of the skin.

Among these should be included shock following severe injuries to the abdomen. In this case the anemia may be due either to a reflex contraction of the vessel of the brain or it may be set up either by a reflex inhibition of the heart or an excessive dilatation of the vessels of the abdomen. A relaxation of the vessels of the abdomen is quite possibly the cause of cerebral anemia which frequently accompanies severe diseases of the alimentary tract.

Chronic anemia of the brain is generally associated with a general anemia or specific disease of the blood (leucemia, pernicious anemia), and it may also be due to increase of the intracranial pressure. In very occasional cases it may be due to compression or thrombosis of the carotid or even stenosis of the aortic orifice.

**Anatomical Changes.** The meninges appear very pale and their vessels in a state of collapse, no plexuses of small vessels being visible. The cortex shows scarcely a trace of pinkish color and the line of demarcation between it and the white matter is indistinct. On section only a very small number of minute points of blood are visible.

**Symptoms.** In severe acute anemia of the brain there is some loss of consciousness from the outset, animals stumble, pigs and dogs vomit and very soon fall to the ground, where they remain as if dead.

The mucous membranes are pale, pupils dilated, papillæ of the optic nerves pale, pulse rapid and thready, respiration irregular—slower and deeper than normal or shallow and rapid. This state of affairs may last for a varying length of time, and there may be general convulsions followed immediately by death (so-called “nervous apoplexy” of old authors). In slight cases there is a gradual return of consciousness, and this may be followed either by complete recovery, or the animals may be weak and dull for some time and for some weeks show a tendency to giddiness.

The symptoms of **chronic anemia** are less striking; besides dulness there may be fright, frequent starting up, spasms of certain groups of muscles and even general convulsions. Owing to the adaptability of the brain, cerebral symptoms may be quite absent in chronic anemia.

**Prognosis.** This depends upon the nature of the cause of the condition. It is a favorable sign if the pupils contract again and react to light. If there are convulsions, severe anemia is indicated.

**Treatment.** Various stimulants may be used, vigorous massage of the surface of the body, smelling of vinegar, oil of mustard, ammonia, subcutaneous injection of ether, caffeine, oil of camphor and tincture of musk. If possible, alcohol, black coffee or brandy should be administered. Artificial respiration if necessary, stimulation of the sensory nerves of the skin, and in small animals faradization of the phrenic nerve are recommended. Finally, injections of physiological salt solution or copious clysters may be given.

In chronic cases the primary disease must be treated, particular attention being paid to the general anemia.

## 6. Injuries and Concussion of the Brain.

**Etiology.** The causes of bruising of the brain are exclusively traumatic injuries involving the cranium or some other part of the head. In the horse, the commonest causes are kicks, collisions, and so on, whilst in the dog it is most frequently caused by falls from a height, blows on the head, or being run over. In ruminants and swine, such cases are less common, but exceptionally concussion may be caused by falls or injuries inflicted with the horns. In birds, traumatism of the brain is in most cases caused by blows on the head during flight.

**Pathogenesis.** Any of these causes may, under certain circumstances, result in depression or fracture of the bones of



the cranium with simultaneous hemorrhage into, and more or less extensive bruising of the brain tissue. Lesions of the skin may be quite trifling or even unrecognizable, and there may be bruising of the brain and membranes without apparent injury to the cranium. At the instant that the injury occurs the brain tissue receives a shock and is bruised or even destroyed by the concussion or the forcing inwards of portions of the bones. There is a sudden rise in the intracranial pressure. As a rule, injuries to the brain and membranes cause symptoms of a general nature, but in a proportion of cases there are local symptoms.

**Anatomical Changes.** Fracture of the bone occurs either at the place where the blow is delivered or at some other part of the cranium. In the horse fracture of the base of the cranium is especially likely to follow a blow on the poll or a fall onto the chin, because the base of the cranium is less resistant, and at the moment of the blow is forced against the rigid cervical vertebræ.

The lesions in the brain vary from case to case, and as a rule are found at the place where the injury has occurred, but in other cases they are in parts of the brain remote from the seat of injury. As a rule the lesions of the intracranial structures can be recognized by hemorrhage of the anterior portion of the brain, base, medulla, or cerebellum and may even extend into the lateral ventricles. In cases where the bones are injured, and even in cases where there is no such injury, a larger or smaller portion of the brain may be reduced to a pulp-like mass mixed with blood.

In many cases there are no lesions visible to the naked eye, in spite of the fact that severe nervous symptoms may have been presented by the animal during life. Such cases are generally included under the term concussion of the brain, it being supposed that the temporary or permanent disturbances of function of the nerve elements are due to molecular alterations of the nerve tissue and not to gross lesions. Experiments by Westphal, Schmaus, Oppenheim and Bikeles, and two observations of Hutyrá & Marek show that in cases where the central nervous system is apparently uninjured there is extensive degeneration of the nerve tissue, and sometimes small hemorrhages showing that there has been some tissue disorganization. There are, therefore, no grounds for distinguishing between concussion and contusion of the brain.

**Symptoms.** Symptoms of general disturbance make their appearance, as a rule, immediately after the infliction of the injury. In other cases, some minutes elapse before their appearance, and it may be even an hour or more. This is especially the case when the disturbances are exclusively or mainly due to hemorrhage. In a case in a horse recorded by Hoffman,



symptoms did not appear until 20 hours after the injury. In this case the brain and membranes were injured by the penetration of a lance head through the lateral wall of the cranium. In quite slight cases the animals stumble and fall, rising again after a longer or shorter time. They may then appear to be quite recovered or they may show local or general cerebral symptoms, either for a time or permanently. On the other hand, in severe cases, the animals go down at once and die within a short time with convulsions and without having regained consciousness. In a moderate case, the animal lies on the ground for some time in a state of complete unconsciousness. The pupils are dilated and the reflexes are quite absent or greatly inhibited. Respiration is slow and irregular, or may be rapid. The heart and pulse are generally accelerated but may be slow and often arrhythmic. Involuntary evacuation of feces and urine are often observed. In pigs and dogs there is often vomiting.

After a time varying from a few minutes to some hours, fibrillar twitchings appear in single muscles or in groups. There may be spasms which increase in violence. Nystagmus is sometimes seen. After a time the animal raises its head and tries to rise, and often succeeds. In cases where there is no direct or indirect injury to the motor paths there may be more or less extensive paralysis and this makes it impossible for the animal to stand or move.

Eicke observed the following symptoms in a dog that had hemorrhage of the brain resulting from a kick from a foal: A great tendency to bite and chase other animals and persons, a stumbling gait and frequent falls, dropping of the lower jaw and foaming at the mouth.

If the animals are conscious to some extent, and the functions of the portion of the brain that is destroyed or injured are well known, local symptoms may be recognized which were previously obscured by the comatose state of the animal.

Hemiplegia is seen comparatively frequently in cases in which there is some destruction of the central motor path. The hemiplegia is either partial or complete on the side of the body opposite to that on which the injury is situated, there often alternating hemiplegia with supra-nuclear (central) paralysis of one half of the body and nuclear or infra-nuclear (peripheral) paralysis of individual cranial nerves on the same side as the lesion. Unilateral paralysis is as a rule, more or less pronounced under certain conditions. There may be paralysis of cranial nerves without hemiplegia. Sometimes both sides of the body are affected by a supra-nuclear paralysis. In one particular case in a dog, the fore legs were paralyzed, the animal pushing the paralyzed anterior part of the body along with the hind legs. Injuries to one half of the cerebellum or the peduncle of the cerebellum may lead to weakness of both limbs on the same side.

Another striking symptom that is not rarely seen in small



animals resulting from bruising of the peduncle of the cerebellum or the posterior portion of the optic thalamus is "waltzing" or rolling or falling over on one side. These movements are also seen in cases of paralysis of some of the cranial nerves, or in cases of obliquity of vision (see page 593). Lateral displacement of the head and neck are seen after injuries involving one hemisphere, the peduncle of the cerebellum, or neighboring tissues. When the head is turned to one side, the animal moves in circles, if movement be possible at all. Many injuries of the hemispheres are followed by epilepsy of the Jacksonian type (see epilepsy).

Hemianesthesia frequently follows concussion of the brain and it is sometimes very difficult to discover even after the disappearance of the coma. Paralysis of the sensory nerves is easily recognized (trigeminus, glosso-pharyngeal, vagus). Concussion is often followed by uni- or bilateral blindness, the exact nature of which has not been ascertained.

After the lapse of a certain length of time, local symptoms may disappear either partially or completely if the affected portion of the brain be subjected only to pressure of extravasated blood in the neighboring tissue, or if it is not completely destroyed.

In many cases, in addition to the nervous symptoms, evidence can be found of the effects of the blow such as wounds, changes in the form of the cranium, increased sensitiveness, etc. When the base of the cranium is fractured there is very often injury to the vessels of the pharynx or ear, and there may be hemorrhage from both nostrils or the ears immediately or soon after the injury is inflicted.

The following are the symptoms of cerebral hemorrhage in **birds**: The head and neck are twisted on the long axis in such a way that the parietal region is directed to one side or even downwards, thus making it difficult or impossible for the bird to take food or water. While walking, they fall down on the slightest provocation, and are seized with convulsions. In many cases, they appear giddy, and stumble, or move in circles, move backwards or suffer severe disturbances of consciousness, and have epileptiform seizures.

**Diagnosis.** Diagnosis is rarely difficult. Difficulty arises in cases in which no injury can be discovered. In such cases, cerebral hemorrhage, embolism of vessels of brain, and encephalitis must be taken into consideration. In the absence of causal factors that are likely to be followed by encephalitis, the sudden appearance of disease unaccompanied by fever indicates an injury. In birds, contagious diseases, such as fowl cholera and chicken pest, must be borne in mind and also the fact that disease of the inner ear in chicken pest, diphtheria, chicken pox and suppurative inflammation of the ear may cause the head to be rotated on its long axis or turned to the side.



Apart from any discoverable lesions of the cranium the localization of the disease must be based upon the local symptoms shown.

**Prognosis.** In the majority of cases concussion of the brain terminates fatally either directly or as a result of complications. Nevertheless cases of recovery are not absolutely rare in both large and small animals. Needless to say, recovery only takes place in those cases in which there is no extensive destruction of brain tissue and no great amount of hemorrhage. The course taken by the disease affords information on this point. Patients which to commence with have shown serious symptoms may survive, but, as a rule, their value is more or less diminished, owing to permanent effects. It sometimes happens in cases of fracture of the base of the cranium that the condition of the patient shows a rapid and striking improvement, but afterwards there is a sudden turn for the worse and the case terminates fatally.

**Treatment.** Complete rest is essential to avoid further hemorrhage, and in animals that are down, the head must be raised. Later, when it is thought that the blood has formed a firm clot in the injured vessels, stimulants, such as ether and camphor, may be given and the animal restored to consciousness by cold applications to the head. Large animals should have plenty of straw under them and they should be turned over from time to time, and if possible placed in slings so as to avoid bedsores. In cases in which the animals remain comatose for a long time nourishment must be administered (see page 123) and the injury to the cranium must be treated surgically.

In view of the doubtful termination of the disease animals that are used for food should be slaughtered as soon as possible if there is not complete recovery in a short time.

**Literature.** Bissauge & Naudin, *Rev. Gén.*, 1905, VI, 212.—Chanier, *Rec.*, 1907, 297.—Dexler, *Nervengerh. d. Pferdes*, 1899, 161, 164 (Lit.).—Eicke, *Pr. Vb.*, 1906, I, 33.—Hoffmann, *Z. f. Vsk.*, 1909, 37.—Joyeux, *Rev. Vét.*, 1906, 525.—Nocard, *A. d'Alf.*, 1882, 681.

## 7. Lightning-stroke.

**Occurrence.** Animals—cattle, sheep and horses—running in the open, are liable to be struck by lightning. Powerful electric shocks act in a manner resembling lightning. Horses are sometimes injured in this way in large towns.

**Pathogenesis.** It is the nervous tissues that suffer principally and there may be pronounced disturbance of function without any obvious injury to the nervous tissues. According to Niedmer these disturbances are to some extent caused by heating of the cerebrospinal fluid.

In the apparently uninjured nervous tissues of men struck by lightning and in animals killed by electric shocks Jellinek found on microscopic examination numerous small hemorrhages scattered through the nerve tissue, patches of nerve tissue that had been destroyed, and in animals that had lived longer, degeneration of nerve fibers.

**Symptoms.** Animals that are struck by lightning may be either killed on the spot or may die in a very short time. In other cases after lying comatose on the ground for some time they get up and may appear quite healthy, or they show nervous symptoms which may persist for a varying length of time or may be permanent. After the disappearance of coma the animals are generally somewhat unsteady on their legs and reel in their gait, the sensorium appearing deadened. As a rule these symptoms disappear in a short time, but not rarely local symptoms persist either for a short time or permanently. Such local symptoms are generally those of paralysis, either monoplegia or paraplegia, or, more rarely, paralysis of particular nerves. There is not uncommonly observed in the horse a permanent or temporary disturbance of vision.

In addition to the lesions of the nervous system there are more frequently seen lesions in other organs, and in particular burning or destruction of the skin and the underlying tissues. On unpigmented parts of the skin the so-called lightning figures can be seen. These take the form of dark-colored streaks or branching lines. This is clearly shown in a preparation made from the skin of a sheep that is in the museum at Munich.

**Course.** There is generally a fairly rapid recovery from disturbances caused by lightning. It is comparatively seldom that animals struck by lightning die at once or later or have to be killed on account of permanent disturbances.

**Treatment.** If the animals do not get up for some time care must be taken that they have plenty of straw to lie upon and are turned from time to time. If there are severe symptoms of sensory disturbance, stimulants may be given (alcohol, ether, camphor, caffeine, stimulation of the skin). As a rule recovery takes place without any treatment whatever.

**Literature.** Dexler, *Nervenkrkh. d. Pferdes*, 1889, 268 (Lit.).—Jellinek, *V. A.*, 1902, CLXX, 56.

## 8. Hemorrhage of the Brain. *Haemorrhagia cerebri.*

(*Apoplexy, Cerebral Apoplexy.*)

Strictly speaking cerebral hemorrhage refers only to such hemorrhages of the brain as are not due to either traumatic influences or inflammation.

**Etiology.** Among the domesticated animals cerebral hemorrhage is due principally to diseased conditions in which there



is mal-nutrition of the vessel walls, and also to acute infectious diseases, such as anthrax, purpura, hemorrhagic septicemia, etc. Hemorrhage of the brain may also occur in cases of anemia, leucemia, hemophilia, chronic hepatitis, and nephritis. In the horse hemorrhage in exceptional cases is due to rupture of an aneurism of some artery of the brain, the aneurism having been caused by the larvæ of sclerostomes (Kitt). In many cases it is due to heart disease (Hering, Scoffie), but neoplasms, such as gliomata and gliosarcomata are rarely concerned, owing to their very infrequent occurrence. So far no one has demonstrated atheromatous arteritis of the vessels of the brain in cases of hemorrhage in the lower animals, and in any case that condition must be far more rare in animals than in man (Dexler). The outbreaks of cerebral hemorrhages observed by Vâth were, in view of the course of the disease, in all probability due to an inflammatory process of some kind.

Diseases of the vessel walls may *per se* lead to hemorrhage or there may be other factors involved. In this connection an increase in blood pressure plays an important part (during work, excitement, etc.).

**Anatomical Changes.** The hemorrhages are, as a rule, small and punctiform, but they may be larger. The majority are to be found in the cortex and the large ganglia. On the cut surface they appear as purple-red points which can neither be scraped off with a knife nor washed away with a stream of water.

Hemorrhages between the membranes, or between the dura and the cranium (intra- or extrameningeal hemorrhage), occur more frequently and especially in cattle. In such cases the hemorrhages tend to be more extensive (see fig. 86).

**Symptoms.** The smaller the hemorrhages and the more localized they are the less is the intracranial pressure increased. Under such circumstances both general and local symptoms are proportionately slight. The capillary hemorrhages that occur in acute infectious and other disease usually pass unnoticed. In cases where the hemorrhages are more extensive the symptoms resemble those seen in cases of contusion of the brain. The animal is giddy, stumbles and trembles, may make involuntary movements, and then collapse and fall to the ground in a state of coma. Death may supervene with convulsions, or there may be recovery sooner or later. In such cases there may be symptoms of a general or local nature which may be permanent or temporary, and these may cause death in the end. If the hemorrhage is not very severe there may be no complete loss of consciousness, the animal only showing giddiness and unsteadiness of gait, and in addition certain local symptoms. Hemorrhages of the medulla which are somewhat more extensive generally



cause sudden death. (Holterbach observed uninterrupted rumination in a cow that was otherwise quite unconscious owing to intermeningeal and intraventricular hemorrhage.)

Judging from the scanty records of the published cases the local symptoms would appear to resemble those seen in confusion of the brain (see page 625).

**Diagnosis.** The sudden occurrence of symptoms of loss of consciousness without any traumatic influence at work and under conditions which suggest the possibility or probability of hemor-

rhage of the brain, indicates that such hemorrhage has taken place either in the brain itself or between its membranes. Hemorrhage may occur in cases of encephalitis but in cases of this kind the loss of consciousness is preceded by some evidence of illness and often by a rise of temperature. The rise of temperature is often observed after the hemorrhage has occurred. Embolism may lead to similar symptoms and this possibility must always be borne in mind in cases in which there is organic disease of the



Fig. 86. Bilateral epidural hemorrhage in a cow.

heart, thrombosis of the anterior aorta or vessels of the head. Sudden collapse may be caused by anemia or edema of the lungs, but in such cases the difficulty of respiration is a prominent symptom. Differential diagnosis is less difficult in cases where collapse is due to heart disease because there is little or no disturbance of consciousness.

As a rule the position of the hemorrhage can only be ascertained by observing the local symptoms during the later stages of the disease.

In herbivora a microscopic examination of the blood is indicated in view of the fact that the case may be one of anthrax.

**Treatment.** As in all cases of hemorrhage rest is of prime importance. Immediately or within a short time after the

hemorrhage venesection should be practiced and a stimulant given (ether or camphor subcutaneously). Cold compresses to the head are indicated. After the disappearance of severe symptoms suitable nourishment that is not too rich may be given, followed by a mild purgative. It is advisable to slaughter animals suitable for food at once if dangerous symptoms are present.

Attempts may be made to cure any paralysis that may be left by systematic passive movements of the affected limb, by massage, and by the application of electrical currents. The systematic administration of potassium iodide is believed to assist in the rapid absorption of the extravasated blood.

**Literature.** Dernbach, Z. f. Vk., 1907, 174.—Dexler, Nervenkrkh. d. Pferdes, 1889, 192 (Lit.).—Eve, Vet. Rec., 1905, 574.—Holterbach, D. t. W., 1909, 288.—Leblanc, J. Vét., 1903, 595.

### Obstruction of the Vessels of the Brain.

#### (*Encephalomalacia.*)

Plugging of the vessels of the brain may be due either to thrombosis or embolism. In cases of embolism there must be some defect in the left half of the heart, or thrombosis of the anterior aorta or carotid. Although such lesions are common in the heart, and especially in dogs, there are scarcely any references in literature to the subject under consideration. It is quite likely that in the future more exact clinical observations and more frequent examinations of brains at the postmortem, may show that the disease is more common. This was an obvious cause of the softening of the brain in a number of the cases recorded. Bouley and Johne found embolism of the cerebral arteries as a result of thrombosis of the carotid in one case; and Vossage records a case of thrombosis of the posterior cerebellar artery in a horse.

In one case there was thrombosis of the great cerebral vein, and in a few cases thrombosis of the venous sinuses of the brain. In a case recorded by Boelmann, there was thrombosis of the great cerebral vein, resulting from a cellulitis of a fore foot that had been in existence for four weeks. There was softening of the white matter and the lesion was confined to the left hemisphere. In a case recorded by Berlin, there was thrombosis of the venous sinuses due to gangrene of the lungs in a horse; while Moussu records one in a cow in which the cause was an abscess of the esophagus. Kitt also records a case of thrombosis of the sinuses in a horse.

Huynen found extensive calcification of the smallest and medium-sized arteries of the membranes in a 10-months-old calf, the vessels of the superficial layers of the cortex being similarly affected around some hemorrhages in the brain substance.

Plugging of the vessels of the brain is followed by degeneration of the nerve elements and neuroglia cells, the brain substance being thus converted into a pulp-like mass (softening of the brain).

It is at present impossible to give a general survey of the



symptoms, as the references to this kind of case are very sparing. It is obvious that the local symptoms will depend upon the portion of the brain affected; and general symptoms will depend upon the local symptoms shown. As in hemorrhage of the brain, thrombosis or embolism of the arteries is responsible for the sudden appearance of general symptoms of brain disease. These may disappear sooner or later, but they may also be responsible for the death of the animal. In cases of thrombosis of the veins or venous sinuses symptoms make their appearance more gradually as in encephalitis.

In Boelmann's case a mare appeared to be dull and its gait was uncertain, the head was drawn round towards the left side, and the animal moved in circles in this direction. At a later stage the animal showed the following symptoms: Paralysis of the left eyelid, partial closure of the left nostril, insensibility of the left half of the head, dullness and later ulceration of the cornea of the left eye, the lower jaw drawn towards the left. Eight days later the animal could no longer stand and death occurred on the 24th day. At the postmortem an old thrombus 1.5 cm. in length was found in the vein of Galen at the point where it enters the sagittal sinus, and another in the neighborhood of the corpus callosum. There was softening of the entire left hemisphere but especially in the middle part.

In a steer suffering from suppuration of the submaxillary gland Moussu observed, two weeks after the commencement of the disease, increased excitability, and eight days later symptoms of excitement alternating with dullness. At the postmortem it was found that the suppuration had extended along the internal carotid and thus reached the base of the brain, and caused inflammation of the venous sinuses followed by thrombosis.

Vosshage saw the following symptoms in a case in which there was thrombosis of the posterior cerebellar artery: Paralysis of the right side of the body, staggering gait and paralysis of the left facial nerve.

In a case in which there was diffuse calcification of the arteries of the brain and membranes Huynen observed coma, elevation of the head, involuntary movements and a staggering gait. The train of symptoms recalled sleepy staggers.

**Literature.** Berlin, Ö. Vj., 1879, LI, 142.—Boelmann, Ann., 1885, 275 (Ref.).—Huynen, *ibid.*, 1907, 80.—Moussu, Rec., 1899, 313.—Schütz, A. f. Tk., 1878, IV, 145.—Vosshage, D. t. W., 1902, 483.

## 10. Encephalitis.

The cause of encephalitis is usually of an infective nature and the diseased areas are circumscribed. There may be centers of suppuration (purulent encephalitis), or there may be non-purulent areas which are red, yellow, or grayish in color and have undergone a process of softening. In some cases there are no areas of inflammation visible to the naked eye (acute non-purulent encephalitis).

It has already been pointed out that in cases of meningitis, the inflammation of the membranes extends to the superficial layers of the cortex, the reason being that the same blood-vessels are common to both. In such cases the inflammation of the cortex is secondary, and as a rule, makes its appearance in the later stages of meningitis. Besides cases of this type, encephalitis occurs in the domesticated animals in which the inflammation involves the cortex only, or if the pia mater in the immediate neighborhood be involved it is only to the very slightest degree. The inflammation of the brain substance is the principal lesion; and the meningitis is not responsible for any observable symptoms.

Distinction between purulent and non-purulent encephalitis, is of



importance from the clinical point of view, as well as from the pathological; since at least in many cases, special methods of treatment may be adopted in cases of the purulent form of the disease.

(a) **Purulent Encephalitis.**

(*Cerebral Abscess.*)

**Occurrence.** The majority of cases occur in young animals and especially in foals, few cases having been recorded in adult horses. Kofler found cerebral abscess in twelve horses out of forty killed on account of staggers. In the other species of animals the disease is very rarely observed.

**Etiology.** Purulent encephalitis generally results from metastasis in diseases in which pyogenic bacteria are circulating in the blood (see page 597). In the horse the majority of cases of cerebral abscess are causally connected with strangles, the vessels of the brain being plugged with emboli composed of infective material. This is supported by the fact that strangles streptococci have repeatedly been demonstrated in the pus. According to Dexler strangles is the cause in about 60 per cent of cases. Purulent encephalitis appears to be of very rare occurrence in other diseases, but it does occur in such conditions as puerperal septicemia (Williams), smallpox (Röll), ulcerative endocarditis, glanders, suppurative pneumonia, pleurisy, and finally pyemia. Trolldenier found a pathogenic streptothrix in cerebral abscesses in a dog (see Vol. I).

Further causes of cerebral abscess are: injuries to the cranium, and the upper part of the parotid region, suppuration or caries of bones near the brain, and in particular the petrous temporal bone in the dog and the middle ear in the pig and birds, acute inflammation of the upper portions of the nasal cavities or the sinuses and the throat, the infective material reaching the brain either along the course of the nerves or the blood vessels. Animal parasites may also be responsible for the condition, the larvæ of the *Cæstrus* and *Cænurus* in the sheep, in cattle larvæ of the *Cæstrus bovis* that have wandered into the cranial cavity, larvæ of the *Gastrophilus* in the horse. Finally, the condition may be caused by the penetration of foreign bodies from the pharynx. Durréhoux found a needle in an abscess of the cerebellum in a pig.

**Pathogenesis.** When a portion of the brain becomes inflamed abscess-formation results from the collection of large numbers of pus cells and a softening of the brain tissue. When there is rapid enlargement of the abscess the intracranial pressure is increased, white corpuscles escape from the vessels in enormous numbers, and the vessels are dilated. As a result of this, and also owing to the effect of the bacterial toxins on

the nerve cells, general symptoms make their appearance, and where certain parts of the brain undergo degeneration there may also be symptoms of a local nature. On the other hand, if the process of abscess-formation is slow, the general symptoms may be quite insignificant or even absent.

**Anatomical Changes.** In cases in which the disease has been in existence for some time the inner surface of the abscess appears to be covered with a finely granular membrane some millimeters in thickness, the so-called "pyogenic membrane." The pus is white or reddish in color and sometimes has an offensive odor. In very exceptional cases the pus appears to be mixed with blood, owing to the rupture of a vessel on the inner surface of the abscess cavity. In the majority of cases the abscesses occur in one of the hemispheres and more rarely in the cerebellum. If they are superficially placed the disease may extend to the membranes and set up suppurative processes there. In deep-seated abscesses rupture may occur in the ventricles. When the lesions are due to metastasis the abscesses are usually more numerous. Johnne found ten and Delamotte and Brochérion fourteen abscesses in the brain. Abscesses caused in other ways are usually solitary and vary in size. In a case recorded by Prümer the cerebellum was converted into a cavity full of pus. Fröhlner records a case in which pus collected in the lateral ventricles as a result of fracture of the frontal bone (pyocephalus).

**Symptoms.** The rapid development of one or more abscesses causes a rise of temperature, provided there is not fever already, owing to some primary disease, and the temperature is subsequently maintained with pronounced variations. As in the case of meningitis there is a rapidly progressive dullness, which at intervals may give place to symptoms of excitement which may amount to mania. In other cases there is great uneasiness from the outset, but exceptionally there may be no evidence of excitement even up to the time of death. Although both the dullness and the excitement tend to be very marked, these cannot be taken as characteristic of suppurative encephalitis. In many cases there are fibrillar contractions and clonic spasms in various parts of the body, and there may be forced movements. Paralysis of various cranial nerves has been observed. Death may take place within a few days or a week or two, the symptoms and especially those of excitement having gradually become more severe.

With the foregoing must be included cases in which the cerebral abscess remains latent for a time and then causes death in one or two days, the animal showing the symptoms already described (Bouchet, Röder, Johnne). Such a course of events is likely to happen in cases in which the process of abscess-formation has proceeded slowly up to a point and then

for some unknown reason becomes rapid and involves the membranes, or bursts into the ventricle. Cases in which the abscess-formation occurs in the frontal lobes of the hemispheres appear to take this slow course.

In the great majority of cases which run a less rapid course symptoms of disease are to be observed for a longer time, and particularly those of dullness or loss of consciousness. Forced movements are seen with comparative frequency, the movement in the majority of cases being in circles and more rarely in other directions.

In these cases there is a tendency for the symptoms of excitement to make their appearance at variable intervals, the animals suffering from attacks of mania, alternating with severe depression. There may also be epileptiform seizures during the periods in which the animals appear to be in perfect health (Lydtin, Trolldenier). Noack had a case of this sort under observation for three months.

Sudden blindness is occasionally observed as a focal symptom. In a case recorded by Bouchet in which there was an abscess in the middle of the left hemisphere and fibrous basilar meningitis, a foal went blind during the night. The right pupil did not react to light at all, while the left reacted for some time. There was total blindness of the right eye, but movements with the hand in front of the left eye were appreciated by the animal for some time. In a case recorded by Thierry, in which there was an abscess in the right hemisphere, there was sudden amaurosis of the left eye. No accurate tests were made regarding the sight of the other eye.

Unilateral paralysis was observed by Haase in a case in which there was an abscess in one hemisphere directly under the membranes. In a case recorded by Greiners a foal sweated profusely after every meal for a period of four months. At the postmortem an abscess the size of a hazelnut was found in the right half of the cerebellum. Shortly before death the animal had shown respiration of the Cheyne-Stokes type. A pig which had an abscess the size of a hazelnut in the right half of the cerebellum staggered in its gait, walked in circles to the right and fell on its right side in such a way that its snout struck the ground first (Kertész). In a case recorded by Haas a cow was observed to carry its head to the left side owing to the presence of an abscess the size of an apple in the left hemisphere. Any attempts to make the animal carry it straight caused attacks of mania. A horse in which there was an abscess in the posterior portion of the vermiform process of the cerebellum showed giddiness and an uncertain gait, and fell frequently (Jacoulet).

In some of the less acute cases there is no elevation of temperature, but it is probable that systematic testing would show a rise in some cases.



**Diagnosis.** This can only be based on the appearance of general or local symptoms indicating that the brain is involved in diseases in which there is a known tendency for pyogenic bacteria to invade the brain. The very acute cases cannot be distinguished from cases of acute meningitis. Cases in which the course of the disease is slower can be differentiated by their longer duration, the striking improvements which occur at times, the absence of sensitiveness over the cranium and the special local symptoms which not rarely make their appearance. The presence of a primary abscess in some part of the body is in itself no proof that the occurrence of cerebral symptoms is due to purulent encephalitis, because meningitis may also occur under the same circumstances. One must also not lose sight of the fact that in many cases of suppurative encephalitis an animal that was previously apparently in perfect health may become seriously ill owing to secondary meningitis and succumb to it in a very short time. In cases of tumor formation in the brain differential diagnosis can always be based on the absolutely negative history of the disease, its slow development, the presence of a primary growth in some other organ, or in neighboring portions of the cranium, and finally from the engorgement of the optic disc. A periodical rise of temperature also indicates suppurative encephalitis.

An accurate analysis of the focal symptoms that may be present in cases that are not very acute renders a localization of the disease possible, but a very careful investigation appears to be necessary in this connection.

**Treatment.** Provided a correct diagnosis has been made regarding the nature and localization of the disease, and this in the present state of knowledge is of rare occurrence, surgical interference may be resorted to. Good results have followed this treatment in the human subject. At the most a favorable result might be expected in the case of an encapsuled abscess superficially placed in a hemisphere; operation appears to be hopeless from the outset in cases where there are multiple abscesses deeply placed.

**Literature.** Cadéac, *J. Vét.*, 1897, 28; 1907, 588.—Dexler, *Nervenkrkh. d. Pferde*, 1899, 200 (Lit.).—Johns, *S. B.*, 1879, 14.—Kotler, *Monh.*, 1903, XIV, 71.—Niebel, *Pr. Mt.*, 1856-57, 122.—Noack, *S. B.*, 1893, 125.—Picard, *Ann.*, 1904, 531.

**(b) Acute Non-purulent Encephalitis. Simple acute encephalitis.**

Acute non-purulent encephalitis results from an infection or intoxication which in many cases is the result of an infectious disease. In the majority of cases there are numerous centers of disease and these may be hemorrhagic in character.

**Historical.** In 1878 a case was recorded in the horse by Friedberger, and this was followed later by cases described by Thomassen,



Montané Desoubry and Nesmeloff. The cases of cerebral apoplexy in cattle recorded by Vâth in 1892 were apparently in reality cases of encephalitis. More recently Dexler (1899, 1903, 1904) described a diffuse hemorrhagic inflammation of the brain substance in cases of so-called "blind staggers" and other diseases in the horse. He has given a very clear account of the disease from a clinical point of view and also of the pathological anatomy. A contagious hemorrhagic encephalitis was observed by Buckley & MacCallum in Maryland (North America) in 1900. Cases of acute encephalitis have also been recorded by Lesbire & Forgeot, also by Marek. The communications by Kolesnikoff, Brusso & Galli-Valerio, Dexler, Nissel, Liénaux, Marek, Marehand, Petit & Coquot, and Pécard, regarding encephalitis in cases of influenza, must be mentioned.

**Occurrence.** In all probability all the domesticated animals are likely to be attacked, although up to the present cases have been recorded in horses, cattle, dogs and sheep. From these records it appears that the horse and the dog are most frequently affected. Even the non-specific form of the disease may sometimes become contagious.

**Etiology.** The nature of the inflammatory processes involving the brain substance indicate that non-purulent encephalitis is certainly due to an infection or a bacterial intoxication (post-infectious encephalitis, Dexler). The infective material cannot always be detected in the brain tissue, although in given cases no doubt exists as to the infectious nature of the disease. This may be due either to the disappearance of the infective material after the inflammation has set in or to the fact that its action is more accentuated on other organs. As already mentioned bacteria occur with great constancy in the brain in infections of a general nature (see page 598).

According to Dexler and others there is not rarely in cases of influenza in the horse a non-purulent, hemorrhagic diffuse encephalitis, similar lesions being simultaneously present in the spinal cord. Encephalitis, as a rule, sets in during an attack of pneumonia or severe catarrhal influenza, but in some cases only after the disappearance of the symptoms of these diseases. The striking symptoms of cerebral disturbance which occur in many outbreaks are often due to an insignificant diffuse encephalitis.

The cause of the inflammation of the brain which in some cases is hemorrhagic and in others resembles influenza encephalitis as described by Dexler in blind staggers and more recently in horses dead of other acute diseases of the brain is not yet known. Here infection of an unknown nature must be accepted. According to Dexler it may be derived from the lungs, intestines, or other organs, and it is not necessary to demonstrate the cause of the condition in the brain, because it may produce its effects only through toxins circulating in the blood or it may meanwhile have disappeared from the diseased tissue.



The most recent investigation indicates that in many cases there are grounds for connecting the disease with influenza, but one cannot generalize regarding this, because the occurrence of diseases that are similar from the point of view of pathological anatomy in the other species—cattle, sheep and dogs—cannot be placed in comparison with it. In the contagious outbreaks recorded by Buckley & MacCallum no cause could be found either culturally or histologically. The cause was also unknown in the cases recorded by Friedberger, Thomassen, Lesbre & Forgeot, Marek, in the horse, Arloing in the ass, Vâth and Hamoir in cattle, Montané, Hamoir, Nesmeloff, Desoubry and Marek in the dog.

In the dog there can be no doubt that the most frequent cause is distemper, the encephalitis appearing at the same time as other symptoms or after their disappearance. Whether the virus of distemper is itself the cause or whether it prepares the ground for a subsequent infection or intoxication is not yet decided, but the probability is that it is the virus itself or some toxin elaborated by it.

The so-called Borna disease should probably in certain cases be considered principally, if not entirely, as a non-hemorrhagic inflammation of the brain and spinal cord.

The non-hemorrhagic encephalitis occurring in rabies has long been known and will not be further referred to here (see Vol. I).

In no single case has the sun been proved to be the cause of encephalitis (sunstroke), but a priori the possibility of such an effect being due to chemical effects produced by the rays is not excluded. In this case, as in many skin diseases, the rays of the sun should be considered rather as predisposing causes.

It is probable that certain foodstuffs exert only a predisposing action. Many authors have considered the leguminosæ as direct causes of encephalitis. As a matter of fact there are no grounds for the belief that many foodstuffs are capable of setting up an inflammatory process in the brain in a manner similar to the skin rashes caused by foods in association with certain determining factors. The presence of certain nervous symptoms is not always demonstrable, because there may be simply functional disturbance. Butler observed a non-hemorrhagic encephalitis in horses by feeding them on damaged rye and claimed to have produced it experimentally.

Overexertion which in many cases is followed by encephalitis is no doubt only a predisposing cause.

**Anatomical Changes.** Lesions may be found in any part of the brain, the hemispheres, basal ganglia, the peduncles, cerebellum, or its peduncles, the medulla, the gray or white matter. In the fore brain it is usually the gray matter that is involved (Dexler). As a rule there are numerous inflammatory centers scattered over the brain, but occasionally only a single center is



to be found. The centers are, as a rule, the size of a pea only (Fig. 87), but exceptionally they may be as large as a hen's egg (Friedberger). The larger ones are softer than the surrounding tissue and sometimes pultaceous in consistency, and their cut surface is, as a rule, somewhat gelatinous and translucent in appearance. Very small lesions that are not hemorrhagic escape recognition with the naked eye, because they are not different in color or consistency. In many cases there is a pronounced tendency to hemorrhage so that the surface of the brain, the walls of the ventricles, or the cut surface appear beset with reddish-brown hemorrhagic spots varying in size from mere points to areas of considerable size (acute hemorrhagic encephalitis). These large hemorrhages are rarely of even color throughout, but have a variegated appearance owing to the presence of small hemorrhages round about them. In the latter stages the centers become yellow in color owing to destruction of the blood pigments. Should the animal survive, a cyst forms owing to the absorption of the detritus, or there

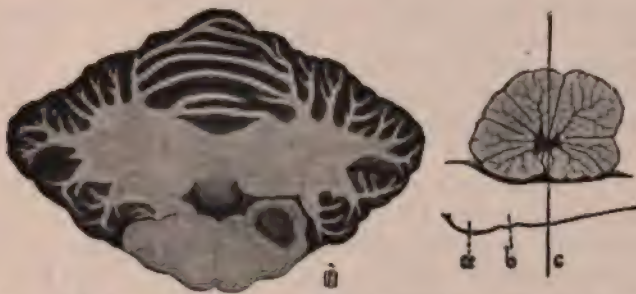


Fig. 87. Area of inflammatory softening of the most anterior portion of the right half of the medulla oblongata in a dog. The animal showed rolling movements to the right. In the diagram on the right a indicates the pons, b the trapezoid body, and c the position of the section.

may be a scar formed. Should the area of inflammation be near the ventricle of the brain, or involve the venous plexuses, a more or less reddish turbid fluid collects in the ventricles (acute hydrocephalus internus). Areas of inflammation near the surface of the brain may lead to the production of extensive or circumscribed patches of meningitis.

The hemorrhagic form of the disease occurs more frequently in the horse than in the other species. Encephalitis due to distemper rarely causes hemorrhages and if present they are unimportant. The hemorrhages found by Brusso & Galli-Valerio were single. Marek has observed a single case of softening of the brain in distemper.

Histological investigations were made by Dexler. Encephalitis in cases of distemper has been investigated by Krawjesky, Brusso, Galli-Valerio, Liénau, and more recently by Marchand, Petit & Coquot, and Pécard. In the cases in which there were no hemorrhages Dexler found perivascular and other cellular in-

filtrations, the cells being round and Marschalko's plasma cells. In one case there was edema of the neighboring tissue. In the hemorrhagic form there was a very small infiltration of leucocytes, but in the perivascular spaces red blood corpuscles were found in large masses and at greater distances from the vessels there were red corpuscles either scattered about or arranged in rows. The endothelial cells had proliferated and were enlarged, the lumen of the vessel being reduced. The true nerve tissue in the neighborhood of the small centers showed very little alteration (destruction of the medullary sheath, swelling of the axis cylinders, and at places destruction of the chromatin bodies of the nerve cells), while the larger centers were composed of softened masses. Degeneration of the medullary sheaths could be followed in both forms of lesion. Not rarely inflammatory lesions were found in the pia mater.

In a portion of cases there was in addition to inflammation of the brain a similar disease of the spinal cord.

**Symptoms.** The symptoms of encephalitis due to any general disease, such as influenza and distemper, are sometimes obscured by those of the primary disease and so may remain quite unrecognized. But in any case of this kind the attention of the observer will be directed to some organic disease of the brain, if by no other symptom, by the pronounced dullness in comparison with the severity of the primary disease.

In the majority of cases the symptoms of brain trouble are very striking. In cases where the inflammatory processes develop rapidly or where the inflammation extends, general symptoms of cerebral disturbance are never absent; these, as a rule, when there is rapid extension, appear suddenly, are very severe, and are ushered in by somewhat severe hemorrhages. They closely correspond with the other acute diseases of the brain; rapidly progressive disturbance of consciousness is a prominent symptom except in cases in which the disease sets in suddenly owing to hemorrhage occurring at the outset. The animals appear dull and listless and are easily fatigued. They are indifferent to their surroundings and stand with their heads dropped or supported upon some object; appetite is greatly decreased or quite absent, and they stand for a long time with half-closed eyes, and their limbs in unusual positions. Occasionally they lose their balance. As a result of this sleepy condition sensibility is decreased. A gradual progression of these symptoms leads, as a rule, within a short time to a condition of semi-consciousness or even coma.

In horses, and more rarely in other animals, the dullness is followed by symptoms of excitement, which may be either very slight or may amount to actual mania. This is especially so in a case of hemorrhagic inflammation in the neighborhood of the ventricles of the brain in a horse described by Dexler. After the period of excitement the dullness is, as a rule, still more marked. Occasionally in cattle there are similar symptoms of excitement, but in the other species, and particularly in dogs, no other symptoms are observed save pronounced restlessness.

Spasms are often observed involving either individual muscles or whole groups of muscles, or they may involve all the voluntary muscles in paroxysms and possibly during the whole



duration of the disease. Forced movements are sometimes observed and they are, as a rule, in the nature of movements either in circles, forwards or backwards.

When the disease is not very extensive, and especially when it involves portions of the brain remote from the cortex, symptoms of a general nature may be insignificant or even absent.

Focal symptoms occur very commonly, but owing to the severe disturbance of consciousness they remain unobserved, or at least they cannot be certainly recognized, save with great difficulty. Paralysis is very common. In some cases it is total hemiplegia (Storch), sometimes hemiplegia alternans (Leisering and Thomassen), and finally there may be paralysis of some of the cranial nerves (ptosis, strabismus, fixed dilatation of the pupils, paralysis of the optic nerves, of the muscles of mastication, the pharynx, tongue and larynx). Extension of the inflammation may lead to a varying amount of paralysis of the whole body with irregular movements of the limbs; dogs can in some cases creep along on their bellies or if supported may be able to walk. Liénaux believes that this kind of disturbance is due to disease of the cerebellum. He observed it in cases of encephalitis due to distemper, associated with exaggeration of the patellar reflexes and nystagmus. It has been shown that it may also follow inflammation involving the cerebrum. When the respiratory center is involved death soon occurs with symptoms of dyspnea.

In the carnivora rolling movements are often observed, and especially in cases of encephalitis due to distemper (Friedberger & Fröhner and Marek). These types of forced movements are often associated with rotation of the head on its long axis, and it is more rarely associated with deviation of one or both eyes. The disease generally involves the peduncles of the cerebellum or the neighboring tissues (see page 593).

In distemper encephalitis after the disappearance of the general spasms, and in some cases without these symptoms having appeared at all, there are observed more or less rhythmic clonic contractions of the muscles supplied by the facial and trigeminal nerves, resulting from a local inflammation in the neighborhood of the nuclei of these nerves.

In cases of extensive disease of the cerebellum, cerebellar ataxia is observed. An especially interesting case of this sort was observed by Marek in a dog, in which there was an extensive perivascular infiltration in the medullary layer of the cerebellum, and at places also in the cortex, the animal being the subject of distemper.

As soon as the dog voluntarily innervates its muscles, trembling movements of that part of the body set in. Thus a nervous shaking of the head was observed in attempts to raise it. In making efforts to stand the animal fell over frequently, while in standing position the toes were extended and great efforts were made to maintain balance; these, however, were more marked during progression. During movement the limbs were swung forward, sometimes with abduction and sometimes with adduction and the feet were put down clumsily, and the animal soon fell either on its side, or forwards or backwards. Every effort to defecate was promptly fol-



lowed by a fall. Movements were further disturbed owing to the eyes being affected. Unnatural attitudes were either not corrected at all or only after a time. The animal was capable of swimming, and only now and then turned on its side so that its head went under water. The eye reflexes were exaggerated up to the death of the animal which occurred more than a month later. There was no demonstrable loss of muscular power.

Loss of sensation in circumscribed areas owing to serious disturbances of consciousness is observed with comparative rarity. Exceptionally amaurosis has been observed (Pr. Mil. Vb., 1897). Dexler saw hyperidrosis in a horse.

There may be a variable amount of fever and in some cases the temperature may be as high as  $41^{\circ}$  C., but in the majority of cases it is not so high, and in protracted cases it may be absent altogether. The pulse varies, as a rule, with the temperature. Excitement causes a slight acceleration of the pulse. From the commencement there is some disturbance of appetite, but after the onset of severe symptoms, and even in cases that are apparently primary the animal ceases to take food altogether.

In many cases in the later stages symptoms of meningitis and myelitis set in. The cases of encephalitis recorded by Buckley and MacCallum closely resemble cerebrospinal meningitis.

The **diffuse encephalitis** seen in cases of distemper, causes a peculiar train of symptoms which was described in detail by Nissl, and afterwards by Dexler, whose observations were made on a number of cases. There is a gradually progressive loss of consciousness associated with pronounced local symptoms and severe motor and sensory disturbances. In the early stages there is dullness, and more rarely a certain amount of restlessness; later, they make no response when called, without showing actual disobedience. They no longer recognize their masters or surroundings, are unable to find places that they are accustomed to, blunder into obstacles, climb over them awkwardly, or remain standing in front of them for hours in a senseless manner, and take up the most uncomfortable attitudes. They lose their power of finding their way about absolutely, and will not attempt to escape through the open door from a room that is unfamiliar to them. If placed upon something at a height, they either make no attempt to jump down or simply fall off. The sense of smell is dulled, animals breathing ammonia without making any efforts to avoid it. Sounds, which under normal conditions produce an active response, are not noticed. The sleepy condition gradually merges into coma, the primary disturbances of the sense organs being thereby obscured. Owing to the fact that animals will not take any nourishment of their own accord, and possibly owing to other unknown causes, there is rapid loss of condition, and death takes place on an average within about one to three months. This condition of dementia should not be termed a psychosis in the true sense of the term; since it is brought about by organic disease of the brain, and is manifested by local symptoms (Dexler).

In a case recorded by Marchand, Basset & Pecard a dog gnawed the lower parts of its hind legs. It would be incorrect to include this among the true forms of mania because a similar condition was set up by Goltz by transverse section of the spinal cord, by Marek by excision of the sciatic nerve, and it is also seen in severe sensory disturbances and even in other cases in which there is no mental disturbance.

**Course.** In the majority of cases the disease lasts only a few days, usually two to five, and in many cases death occurs in even a shorter time, while in rare cases the animal may survive for weeks or even months.

In cases in which the disturbance of consciousness sets in rapidly and in severe form there are frequently focal symptoms also. Both the general and local symptoms increase in severity as the inflammation extends and the local symptoms may also increase in number and their distribution may become more extensive. In such cases death is not long delayed. The general cerebral symptoms and the local symptoms may decrease, or, except in the early stages, disappear. Such a course indicates disease either of the crura or the cerebellum. Obviously such symptoms may cause death in a short time owing to some serious complication or to some portion of the brain essential to life becoming involved. In the few animals that recover there is a tendency to relapses, and according to Dexler this is especially the case in animals that have had hemorrhagic inflammation in the neighborhood of the lateral ventricles. In such cases there is a great likelihood of the recurrence of attacks of mania and staggers.

Even if a relapse does not occur the animals, as a rule, show permanent sensory disturbances. Evidence of the occurrence of such cases of encephalitis should be found more frequently, and especially in horses that have suffered from staggers and at the postmortem of which nothing can be found to account for the staggers. In dogs and in other animals there may persist a tendency to epileptiform fits. In rare cases there are persistent local symptoms which depreciate the value of the animals. There is no doubt that in certain cases of encephalitis there is complete recovery and this possibly explains the complete disappearance of the severe symptoms observed in some animals affected with distemper or influenza.

In the sub-acute forms there is, as a rule, a gradual loss of consciousness, and the cases terminate fatally owing to the fact that the animals take little nourishment. In the horse there may be relapses of the inflammation from time to time.

**Diagnosis.** If general, and what is of more importance, local symptoms of one of the diseases mentioned in the paragraph devoted to etiology are present, and there is no pain of the cranium, diagnosis is easy. In dogs encephalitis due to distemper may be diagnosed if there are rhythmic spasms of the same groups of muscles. In cases that appear to be uncomplicated the appearance of cerebral symptoms associated with a rise of temperature, acceleration of pulse, and absence of pain over the cranium, raises the suspicion that they are simple cases of encephalitis; suppurative encephalitis is excluded if no primary suppuration can be found in any part of the body and no injury to the cranium can be found. The disease is easily con-



fused with meningitis if the cranium becomes painful on pressure owing to simultaneous inflammation of the membranes, and certain symptoms which contraindicate meningitis (hemiplegia, rolling, cerebellar ataxia) are not present. It is impossible to exclude basilar meningitis with symptoms of a more general nature, or meningitis involving the neighborhood of the ventricles (meningitis interna). Hemorrhage or embolism are indicated by the sudden onset of severe symptoms. Such cases may be excluded by careful investigation of the history of the case, the condition of the other organs, and the temperature. The disease is distinguished from chronic dropsy of the ventricles by the fact that severe cerebral symptoms develop with comparative rapidity, and by the presence of local symptoms. Sometimes it is necessary to distinguish the disease from acute uremia.

**Treatment.** Directions given in connection with meningitis (see page 604) are applicable, but, as a rule, treatment is without avail.

**Literature.** Dexler, *Ergebn. d. Path.*, 1896, III, 2, Abt., 508 (Lit.); 1900, VII, 483 (Lit.); *Nervenkrkh. d. Pferdes*, 1899, 103 (Lit.); *Monatschr. f. Psych. u. Neurol.*, 1903, 97 (Lit.); 1904, 99 (Lit.).—Fröhner, *Monh.*, 1908, XIX, 133.—Lesbre & Forgeot, *J. Vét.*, 1902, 157.—Liénaux, *Ann.*, 1900, 487.—Marchand, *Bas-set & Pécard, Rec.*, 1906, 813.—Marchand, Petit & Coquot, *ibid.*, 1905, 419.—Marchand, Petit & Pécard, *ibid.*, 1907, 357.

**Chronic Encephalitis.** Very few references are to be found in literature to the occurrence of lesions apart from chronic meningitis. These take the form of white or grayish translucent firm nodules in the brain which, to all appearances, have been caused by an acute encephalitis. Buckley and MacCallum found sclerotic nodules in the brain of a horse that had recovered from a hemorrhagic inflammation of the brain, and had afterwards shown symptoms of staggers. Lellmann concludes from some observations made by himself that multiple sclerosis of the brain occurs as frequently in animals as in man, but it is only an assumption without any anatomical proof. Finally in a dog that had recovered from distemper, Meissner found a bladder-like swelling and thinning of the dura mater over the right hemisphere in the region of the parietal bone, a flattening of the convolutions, and on the mesial surface of the hemisphere, a cavity extending to the ventricle (porencephalia). During life there was a certain amount of awkwardness, lack of intelligence, bilateral amaurosis and occasional attacks of cramp and movement to the left.

## 11. Chronic Dropsy of the Ventricles. Hydrocephalus internus chronicus.

(*Chronic Hydrocephalus; Dumbness.*)

By this term is indicated a chronic disease of the brain characterized by the presence of abnormally large quantities of cerebrospinal fluid in the ventricles of the brain with consequent



dilatation of these cavities, an increase in the size of the brain, and an elevation of the cranial pressure. The disease may be primary or secondary to some other disease of the brain.

**Occurrence.** The disease is seen most frequently in the horse in which animal it is the commonest cause of sleepy staggers. Up to the present there is no very exact information as to its frequency. Having regard to the anatomical observations made by Dexler, it remains to make investigations to decide how often the condition is the cause of staggers, and how often it is the result of some other diseased condition of the brain. No stress can be laid on the results obtained in the past because no comparison was made with regard to the anatomical alterations found, and measurement of the size of the ventricles allows of a great chance of error.

There are some cases on record of dropsy of the ventricles in the dog (Fröhner saw 20 cases among 70,000 dogs), the pig (Schindelka), and also in other species, but either the true nature of the disease is not represented, or no proof has been adduced that the cases are actually cases of chronic internal hydrocephalus. In many cases the possibility of confusion with other chronic brain lesions not recognizable with the naked eye is not excluded.

**Etiology.** The lateral ventricles of the brain communicate with the third ventricle through the foramen of Monro, and this is in communication with the fourth ventricle by means of the aqueduct of Sylvius. As shown by Dexler the cerebrospinal fluid may reach the sub-arachnoidal space from the fourth ventricle by way of the two lateral foramina, and here be reabsorbed by the veins. Cerebrospinal fluid may collect in the ventricles first if an abnormally large amount of serum escapes from the venous plexuses in the ventricles and cannot pass freely through the relatively small aqueduct, or when the outflow of the otherwise normal amount of liquid is prevented owing to a narrowing or obstruction of any of the paths of communication. Dexler was the first to supply a satisfactory explanation of the collection of the cerebrospinal fluid. According to this the immediate cause of chronic internal hydrocephalus is stenosis or occlusion of the aqueducts of the brain.

Dexler's investigations have shown that the tentorium cerebelli in the horse is for the most part ossified and that its membranous part is composed of very tough connective tissue. The tentorium encloses a space measuring 4.2 cm. in height and 3.5 cm. in width, allowing for individual variations, and terminates above in an angle. This space is occupied by a very small portion of the vermiform process of the cerebellum and the corpora quadrigemina together with the aqueduct of Sylvius and the crura cerebri. The oral surface of the tentorium cerebelli lies behind the convex surfaces of both occipital lobes of the hemispheres; the medial surfaces of these coming into contact with each other. In view of the fact that the brain is enclosed in a bony case and that the brain tissue which contains a large amount of fluid is incompressible, pulsation of the brain at each systole is only possible by the expansion of the brain at the systole into the subarachnoid space, the ventricles, and into the cavity of the tentorium cerebelli. Since the tentorium is very rigid, pulsation of the brain in the horse is conveyed only to a comparatively small portion of the brain lying behind the tentorium. Consequently the portions of the occipital lobes resting on the walls of the tentorial cavity are pressed into the open-

ing at each heart beat and in time may form a three cornered protrusion 2 mm. in height. This often occurs in normal adult horses when adhesions may form with the part on the opposite side.

In the other domesticated animals the anatomical relations differ in important details from those obtaining in the horse. In the dog the falx cerebri completely separates the posterior portions of the hemispheres, the tentorium cerebelli is completely ossified, and its edges closely cover the anterior pair of corpora quadrigemina. In ruminants the medial surfaces of the hemispheres are in close contact throughout their extent, the tentorium is purely fibrous and has a very wide opening which allows the anterior third of the cerebellum to extend into the larger cranial cavity, and the posterior portions of the hemispheres cover the cerebellum to a considerable extent. In the pig the opening of the tentorium is somewhat smaller than in cattle but it is sufficiently large to allow of somewhat extensive contact between the cerebellum and the hemispheres. While in the dog the separation of the cerebrum from the cerebellum is complete making protrusion impossible, protrusion is prevented in ruminants and pigs owing to the fact that the space between the cerebrum and cerebellum is too great. Swelling due to pressure (protrusion) is possible then only in horses. From the anatomical point of view the horse occupies the middle position.

Under the action of repeated or long-continued pulsation of the brain, or increased intracranial pressure, a large portion of the occipital lobe is forced through the tentorial opening and this causes more or less pronounced pressure upon the corpora quadrigemina lying below. The compressed corpora assist in the compression of the aqueduct, thus hindering the outflow of the fluid from the ventricles (lateral) into the fourth ventricle and thence into the sub-arachnoid spaces. Once this passage is obstructed the forced collection of cerebral fluid in the ventricles causes a rise in the intracranial pressure and this causes greater protrusion of the occipital lobes and still further compression of the aqueduct. Once the process is started recovery is impossible, the condition becomes aggravated with intermissions, and gradually in most cases leads to complete occlusion of the aqueduct of Sylvius.

All factors which cause a rise of intracranial pressure either for any length of time or repeatedly may set up chronic dropsy of the ventricles. In this connection special mention should be made of acute meningitis, frequently repeated or persistent cerebral hyperemia, acute encephalitis, tumors of the brain, etc.

Not rarely the disease appears to be primary and makes its appearance without any of the above-mentioned diseases being in existence (primary internal hydrocephalus). In such cases the excessive pulsation of the brain is due to various sensory impressions, excessive work, labored respiration, variations of temperature, poisons, etc. As a rule no increase of blood pressure can be demonstrated.

In no instance in his accurate investigations did Dexler find any histological alterations in the venous plexuses, choroid plexus or ependyma lining the ventricles. He therefore thinks it probable that the normal quantity of fluid escapes from the plexuses and that the congestion is not therefore produced by an excess of fluid escaping from the veins. If this were true, as cannot be shown in cases following acute inflammatory processes, there would be no pronounced protrusion of the occipital lobes or compression of the aqueduct.

Rarely the disease may be set up in other ways. The foramen of Monro may be occluded by parasites (echinococci, cœnuri), tumors in the neighboring tissues, closure of the lateral foramina owing to chronic meningitis. Possibly cases of this sort occur in other animals.

**Cholesteatomata\*** which are found comparatively frequently in connection with the choroid plexus in the horse probably are not causally connected with hydrocephalus internus because they are as often absent in animals so diseased and present in otherwise healthy horses as in animals that are affected with dropsy of the ventricles. In exceptional cases the ventricles even when cholesteatomata are present as large as a nut or larger do not contain more liquid than normal.

**Predisposition.** It is generally supposed that a predisposition is hereditary. As a matter of fact experience shows that thoroughbreds and racing breeds are rarely affected with sleepy staggers, whereas heavy breeds are often so affected. It appears to be not impossible that in heavy breeds the anatomical formation of the cranium is less favorable (small cranium with relatively large tentorial opening), permitting greater protrusion of the occipital lobes or exerting a prejudicial influence on the pulsation of the brain. In view of the fact that the cranial formation is a hereditary feature, predisposition may also be transmitted from generation to generation. The fact should not be lost sight of that the heavy breeds do the heaviest work and consequently are more likely to suffer from repeated elevations of intracranial pressure. There is no recorded evidence of the hereditary nature of the disease.

In support of the hereditary theory it is stated that staggers is of so common occurrence in many districts and particularly in mountainous districts that horse-breeding is impossible (Trasbot). The frequent occurrence of the disease may be connected with local conditions such as chronic poisoning with bad food or it may be connected with repeated infections which set up chronic diseases of the brain.

As a rule it is mares and geldings that are used for work that suffer most. Trasbot saw the disease principally among stallions.

Age plays some part in the production of the disease. The majority of cases occur in horses from six to fourteen years old. The disease seldom occurs before that, and in animals under two years old it has not been observed. The disease has been connected with the changing of the teeth, but this cannot play any part in its production.

The occurrence of hydrocephalus ex vacuo has not as yet been observed in animals, because animals rarely reach such an age that senile atrophy of the brain tissue would lead to dilatation of the ventricles.

**Anatomical Changes.** The lesions produced by the presence of liquid under pressure in the ventricles depend upon the severity and duration of the disease. These may in part disappear when the pressure is relieved, but they may persist up

\*See footnote, page 656.



to the time of death. The quantity of liquid in the ventricles in cases of hydrocephalus may be from 40 to 120 grams (Hering). Dexler was able in a single case only to show increased pressure in the cranial cavity after death.

According to Dexler there is in the first place dilatation of the lateral ventricles and of the anterior part of the third ventricle. As a result of this the brain, the weight of which is found to be normal after the escape of the liquid, is enlarged, and the liquid passes into the lymph spaces of the cranium and especially into those of crura cerebri, the pituitary body, the decussation of the optic nerves and the fossa of Sylvius. In view of the fact that the brain is enclosed in a bony case the dilatation of the lateral ventricles cannot be very great. The



Fig. 88. Chronic hydrocephalus internus. Section posterior to the hemispheres. a. Swelling formed by the medial portion of the occipital lobes (c); b. Posterior border of the flattened corpora quadrigemina; d. Aqueduct of Sylvius reduced in caliber to a fissure-like opening. e. Optic chiasma.

dilatation is most pronounced in the middle portion of the ventricle and in the vertical direction, and least in the inferior horn, because in healthy horses there is often some adhesion here and this prevents dilatation. The olfactory bulbs are sometimes dilated to a certain extent. The septum pellucidum between the two lateral ventricles is stretched and sometimes perforated. The posterior part of the third ventricle appears reduced in size owing to the bulging of the corpus mammillare into the lumen. In consequence of this the aqueduct appears to be either greatly reduced in caliber or even completely closed. On the other hand, the anterior third of the ventricle appears to be broader, the recessus infundibuli occluded owing to the

bulging of the pituitary body and the optic recess is usually enlarged. In severe cases the middle portions of the optic thalami, the optic decussation and both the optic tracts appear to be flattened. The portion of the epiphysis above and in front of the conarium is sometimes dilated.

On the medial surface of the occipital lobes there is a triangular protrusion, the size of which depends upon the extent of the disease; this protrusion generally shows normal convolutions (Fig. 88a). The depressions appear shallowest where the two prominences are in contact. The enlargement of the occipital lobes already mentioned exerts pressure in proportion to the enlargement in the backward direction on the corpora quadrigemina, forcing the anterior pair apart, and presses them against the base of the brain. The enlarged occipital lobes also exert pressure with their lateral surfaces on the corpora quadrigemina towards the middle line, thus flattening them and forming a saddlelike depression on the anterior pair (Fig. 88b).

The aqueduct appears to be reduced in caliber not only on account of the compression of the posterior part of the third ventricle, but also owing to the pressure by the corpora quadrigemina, with out there being any adhesion of its walls. The cerebellum is pushed further back, the surface of the crura cerebri is smooth and not cordlike, the oculomotor nerve appears to run a longer course and is pressed flat (Fig. 89). The anterior border of the pons is sometimes curved in the upward direction.

In a portion of cases there is gelatinous infiltration of the venous plexuses, formation of cysts with delicate walls, cholesteatomata\* and thickening of the ependyma.

In an acute relapse there are small hemorrhages under the ependyma, and there may be even centers of softening. The ventricles frequently contain a turbid liquid which in some cases

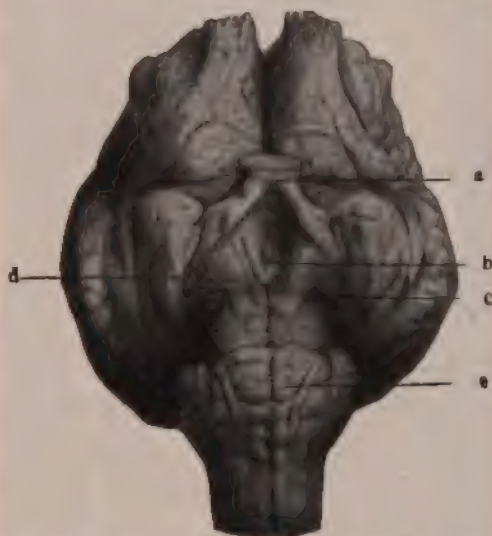


Fig. 89. Chronic hydrocephalus internus. View of the base of the brain shown in fig. 88. a. Optic chiasma. b. Corpus mamillare, forced backwards, wrinkled and showing a depression. c. Crus cerebri elongated and flattened and showing an oblique furrow. d. Oculomotor nerve, flattened and stretched. e. Pons Varolii, wider than normal and with its anterior border curving forwards.

\*See footnote, page 656.



contains flocculi of fibrin, the latter being the result of meningo-encephalitis.

**Symptoms.** All the symptoms of the disease are referable to the pressure which affects all portions of the brain anterior to the tentorium cerebelli, the hemispheres and the large basal ganglia. Consequently general cerebral symptoms are observed to be most prominent, but there are also disturbances of the body functions.



Fig. 90. Horse with internal hydrocephalus.  
("Dummy.")

The disturbances of consciousness cause, above all, a change in the demeanor of the animals (Fig. 90). The animal appears to be more or less indifferent to its surroundings and stands in a sleepy condition with head sunk, half-closed eyes and a vacant expression. The head is frequently rested upon some object, there is either little or no movement of the ears and the animal does not always turn them towards a sound, but frequently in the opposite direction (so-called reversed ear movements). Faint sounds usually call forth no reaction on the part of the animal, but louder noises, such as the banging of a door or clapping the hands, make the animal collect itself, but it soon relapses into the sleepy state (narcolepsy).

Appetite is variable; the animal may take hay or straw into its mouth, but allows it to hang out between the lips. Both eating and drinking appear to be abnormal; the animal buries its widely opened mouth quickly into the food, gets too large a quantity into the mouth and chews it slowly. While drinking the head is lowered down until the nostrils are covered by the water and it is soon withdrawn suddenly owing to difficulty of respiration. In many cases the horses make movements as if



eating while drinking or forget to swallow. Some animals consume their usual diet, others eat sufficiently only to allay hunger. In many cases animals prefer to eat off the ground or out of the manger rather than from the rack. This is not because the pressure varies with different positions of the head, because the same or a similar tendency is seen in other chronic diseases of the brain in which there is no increase in the intracranial pressure.

Sensibility appears, as a rule, to be decreased. Affected animals take no notice of slight pressure on the skin, do not attempt to remove flies, remain more or less quiet if pricked with a needle, the coronet be trodden upon, the flank pinched, the hairs around the muzzle be pulled or the ears seized. The sense of perception of position is sometimes in abeyance, the legs being placed in quite unnatural positions, sometimes widely separated and at other times crossed. If the animal be placed in an unnatural position it will remain in it for a long time until some disturbance of balance or other stimulus causes it to correct it.

Abnormalities of movements are shown by the reluctance with which an animal moves when urged to do so. In some cases the animals are restive, making sudden plunges. It is particularly difficult to move a horse backwards, the forelegs are moved with this object, but the hind feet remain stationary until movement is forced owing to loss of balance. While walking or trotting the feet are lifted unusually high and are put down clumsily, as if the animal were walking through water. If the ground be uneven the head is held either very low or excessively high.

Skin reflexes are, as a rule, abolished, but sometimes they are exaggerated. In some cases (Hutyra & Marek) the patellar reflex was exaggerated to a marked degree.

Abnormal movements are not actually rare and in the majority of cases the animal has a tendency to go towards one side, in contradistinction to moving in circles. The head in such cases is often held obliquely.

The sense of vision is sometimes disturbed and the evidence of fright, which is not infrequently seen, is probably due to this. In severe cases the sense of vision may be quite lost, or there may be complete amaurosis. Lustig records venous congestion of the optic papillæ and marked redness round about them; others, however, have not observed this and Hutyra and Marek have not been able to confirm this.

It is very probable that the other organs of sense suffer some disturbance of function. There are great difficulties in the way of ascertaining the existence of such anomalies in animals.

The heart action is slower and in the horse may fall to thirty to twenty per minute, and it may also be arrhythmic, but it is full and soft. Respiration is also slower, nine and seven

per minute, and the rhythm and type of respiration vary. Movements of the intestines are sluggish; there is some constipation and what feces are passed are dry and in the form of small balls.

The above-described symptoms are, of course, present only when the disease has reached an advanced stage, and even then one or other may be absent. In the early stages the abnormalities are very slight and it requires very careful examination of the animal to discover them. Regular work which has been learned through its having been done for a long time is almost as much an anatomical function as eating and drinking, and for the most part it may be carried out without voluntary impulses and consequently it may be done passably well in spite of the withdrawal of the cerebral influence. Disturbances of the last-named function are noticed much later, the better bred the animal, the more active its temperament and the more thoroughly it is accustomed to its work. The person riding a horse gets the earliest intimation of disturbance of the functions, in that the horse does not carry out certain movements with its accustomed precision (jumping, etc.); difficulty is experienced in making it change its pace and it makes unnecessary movements. Draft horses are, as a rule, not observed to be ill until there are pronounced symptoms.

The symptoms presented by an animal are likely to vary considerably. A sudden rise of the intracranial pressure or an exacerbation of the inflammation, which often occurs in cases of dropsy of the ventricles, may cause a transitory aggravation of the animal's condition. In this connection work is of the utmost importance, for if the animal be worked until it breaks out in a sweat the symptoms which up to this time may have been very indefinite are aggravated to such an extent that the animal may now show the typical symptoms. A similar but less important effect may be produced by very hot weather, warm damp stable, œstrum, rich diet, etc. The opposite conditions are conducive to an improvement in the animal and in the early stages may cause a temporary disappearance of all symptoms.

The effect of external influences upon the symptoms explains the experience commonly met with in practice, namely, that when a horse that is affected with sleepy staggers changes hands it may show symptoms under its new conditions that had not been observed by the previous owner.

There has been observed a suddenly occurring exacerbation of the condition associated with fits, and in many cases the latent disease is first made evident by such an attack (so-called mad staggers), or it may be started in this way (Dexler). The animal does not answer to the reins, cannot be moved, or it takes fright without any reason at well-known objects or even a light. It attempts to break through, or even does break through by sheer force, objects standing in its path. If the seizure occurs while the animal is in the stable it rears up, at-

tempts to free itself and falls over backwards. After an attack which at the most lasts for an hour or two there is a period of depression and great dullness, which gradually passes off after some hours.

The acute attacks which are observed in cases in which the hydrocephalus is preceded by meningitis or encephalitis are of much greater importance. These attacks cause an exacerbation of the inflammatory processes (see pages 600 and 640), and not rarely cause death, although the condition of the animal is not dangerous.

After the passing of these acute attacks, which sometimes last for several days, there is almost always a turn for the worse.

The symptoms above described are characteristic in a general way of the disease known as staggers which is most frequently caused by dropsy of the ventricles. But staggers should not be identified with chronic hydrocephalus because this has a far wider significance than chronic dropsy of the ventricles. Similar symptoms are observed in all diseases of the brain which are of a chronic nature and which are associated with a rise in the intracranial pressure, or destruction of the brain substance (neoplasms, parasites in the cranial cavity, exostoses on the inner surface of the cranial bones, chronic inflammation of the brain, etc.).

**Course.** Chronic dropsy of the ventricles of the brain runs a slow course and apart from the occasional exacerbations there is no fever from first to last. Owing to the accumulations of the fluid and to the consequent rise of intracranial pressure certain symptoms make their appearance which at the outset are scarcely recognizable and which scarcely affect the animal's condition, but which in the course of time become so severe that the animal can be used for rough work only. In some cases the slow evolution of the disease is interrupted by marked improvements and by relapses from time to time. In addition to the factors mentioned above, the season exerts an influence on the disease, there being an improvement in the winter, the animal becoming worse in the summer. It is very exceptional for the disease to progress so far that the animal is absolutely without intelligence, takes scarcely any food, and as a result falls off greatly in condition. If the animal does not die during an acute exacerbation or from some other concurrent acute disease it is usually slaughtered before it becomes absolutely senseless on account of its very limited use.

**Diagnosis.** In the advanced stages of the disease the symptoms due to disturbance of the cerebral functions are so obvious that they can be recognized in an instant. In the early stages their recognition is associated with great difficulty and a systematic examination of the horse is essential. Skill is very necessary in this, because a practiced eye at once suspects loss of intelligence from the general facial expression and particularly from the appearance of the eye, even when there are no abnormalities of movement, sensitiveness or reflexes. The age,



breed, condition and the evidence of wear and tear of the animal must be taken into consideration. It appears to be most useful to have the animal put to do some fatiguing work, because the somewhat indefinite symptoms, as a rule, tend to become accentuated shortly after and also because fresh ones may make their appearance. The animal should be watched the whole time that it is at work, special notice being taken as to whether it responds to instructions. After being allowed to stand in for a time a fresh searching examination should be made.

Points that are of special importance are: unnatural position of the feet, supporting the head on the manger, leaning up against the wall, the periods of rest during feeding. Abnormalities of sensibility have a very limited importance because they are very variable from animal to animal. Above all, a diagnosis should never be based upon the presence of individual symptoms; several symptoms must be present before a diagnosis can be made. Further, it must be remembered that the sum total of the symptoms described is not absolutely pathognomic of the disease, but they may be met with in other diseases in which there is great dullness.

In examinations carried out on 372 sound army horses Dröge showed that quite sound horses will keep their legs crossed, but especially when fatigued or during very hot weather. A positive result was obtained by Dröge in 11% of animals in winter and 30% in summer.

All acute, and particularly febrile, diseases must be excluded, although these are frequently associated with depression of the functions of the brain. The elevation of the internal temperature and acceleration of the pulse, the presence of symptoms of disease of some other organ and the history of the case are, as a rule, sufficient to prevent one from making an error. Inflammatory conditions of the bones of the face and their sinuses are accompanied by great dullness in many cases, but the local symptoms (discharge from the nose and pain) are sufficient to explain this. The disease can still less be confounded with loss of liveliness during the changing of the teeth or sexual excitement. The short duration of these conditions is enough to differentiate them. Chronic diseases of the stomach and liver which are now and then accompanied by depression are easily excluded on account of the systematic symptoms present.

The acute attacks seen in hydrocephalus may be easily confused with acute inflammation of the brain or its membranes. In these cases symptoms of excitement alternate with symptoms of depression as in the acute seizures in dropsy of the ventricles. Differential diagnosis must be based upon the history of the case and upon any local symptoms that may be present. If the case is one of an acute seizure resulting from dropsy of the ventricles, certain disturbances of function will have been present before, while fever, paralysis of cranial nerves, and particularly spasms of the neck muscles, indicate meningitis.

The above diseases having been excluded it remains to determine whether the cerebral disturbances are not the result of some other chronic disease of the brain. As a rule it is very difficult, if not impossible, to ascertain what is the primary disease, but the abnormal movements, the acute attacks occurring at long intervals and lasting for some days, the alternation of improvement with relapse generally indicate internal dropsy of the brain. Paralysis of individual cranial nerves and the exhibition of symptoms on the part of the opposite side of the body indicate either the presence of tumors or parasites in the brain. Finally in cases of hydrocephalus internus only is improvement observed under treatment with diaphoretics. The differential diagnosis of chronic encephalitis and meningitis is fraught with still greater difficulty because these conditions sometimes lead to a gradual depression of cerebral activity.

**Prognosis.** When once the disease has started it pursues a steady course, gradually increasing in severity; however, there may be periods during which the disease, although still present, is at a standstill. Prognosis is very unfavorable, although the disease does not prevent the horse from doing certain kinds of work. The more pronounced the dullness, the more serious are the disturbances of power of locomotion and the more limited the usefulness of the animal. Should symptoms appear indicating that the nutrition of the body is upset it is no longer worth while to keep the animal alive. Riding horses are most depreciated by the disease, because greater demands are made of them as to accuracy of movement, etc. Diseased horses are consequently fit for draught work only, and that at a walking pace. In judging individual cases, besides the diseased conditions, the following points must be considered: for what kind of work is the horse used, under what conditions does it work, and whether it is possible to do away with any prejudicial conditions of its work. Finally, in forming a prognosis the possibility must be considered that acute attacks may occur and cause death within a short time or make the animal dangerous.

**Treatment.** When once the disease has set in treatment is of no avail, but by careful regulation of the diet the progress of the disease may be rendered slower and thus the animal may remain capable of work for a longer time. Above all, diseased horses should be spared as much as possible and used for light work. Food should be given in sufficient quantity to maintain the condition of the body and allow the work to be done. It is not advisable, as many believe, to keep the animals hungry. The food should be easily digested so that constipation may be avoided. Fresh green food is the best in the summer. Finally, the patients should be placed in clean stalls that are not too warm.

In cases in which there is impaction of the intestine medicinal treatment may be resorted to and it is best to give large doses of neutral salts, although now and then aloes may be used with advantage. In cases where there is loss of consciousness two or three subcutaneous injections of pilocarpin hydrochloride (0.30-0.50 g.) or arecolin (0.05-0.10 g.) give some relief. Viborg and later Dieckerhoff advised tincture of veratrine. Six to eight grams were injected intravenously and as long as there was difficulty of respiration, sweating and nausea the animals were left loose in a large box. In acute attacks the same treatment as used in acute meningitis is indicated (see page 604).

Hayne advised puncture of the olfactory bulbs so as to allow the liquid to escape. This treatment is dangerous as it may lead to a fatal meningitis and produces no lasting results (Hering, Röhl, Dieckerhoff).

**Literature.** Dexler, Z. f. Tm., 1899, 242 (Lit.).—Dröge, Z. f. Tk., 1907, 496.—Meissner, B. t. W., 1899, 239.—Schindelka, Ö. Z. f. Tk., 1891, IV, 106.

**Congenital Hydrocephalus.** (Hydrocephalus internus congenitus). This developmental anomaly is equally common in foals, calves, lambs, and dogs, and the condition is more pronounced than the acquired disease. In very pronounced cases the brain appears to be enclosed in a thin-walled sack, which is filled with a clear or slightly turbid serous liquid, the medullary substance forming a thin layer inside the wall. Since during fetal life the bones of the cranium are not yet joined; the cranium enlarges enormously, the parietal and frontal bones bulge, and the orbits are reduced in size, and the head assumes a characteristic shape, the cranium being large out of all comparison with the rest of the head. Such a head may lead to dystokia. As a rule, the animals die soon after birth, but they sometimes remain alive; and apart from the abnormal shape of the cranium, show no symptoms of disease, although the cortex of the brain may be almost entirely absent. (Forgeot & Nicolas, Bull. de la Soc. des Sci. Vét. de Lyon, 1906, 115.—Jellinek, T. Z., 1907, 435.—Marchand & Petit, Bull., 1907, 261.)

## 12. Tumors of the Brain.

**Occurrence.** Tumors within the cranium are of very rare occurrence. In the brain itself the so-called cholesteatoma\* is comparatively frequently found. Dexler found them in 22.5 per cent of 204 horses examined. They develop in connection with the venous plexuses beneath the cerebellum or in the lateral ventricles. The majority are about the size of a pea, but in some cases they attain the size of hen's or goose's eggs. As a rule they cause no disturbance of health. By investigations with the polarization microscope Schmay showed that cholesteatomata present the characters of chronic granulating inflammation and has termed them granuloma cholestrinicum.

\* (The type of neoplasm referred to by continental authors as cholesteatoma is termed, in English veterinary literature, psammoma, the name cholesteatome being reserved for a growth of a totally different nature which is very occasionally met with in the substance of the cerebral hemispheres.—Translator's Note.)



Glomata and gliosarcomata occur as ill-defined reddish growths. Sarcomata and, in the horse, melanotic sarcomata occur in the actual brain substance, the growths being metastatic. The same is true for carcinomata. In cases recorded by Trasbot and Holterbach the primary growth was in the testicle or the mammary gland. Isolated or multiple tuberculous growths occur in the brain substance in a manner resembling true neoplasms.

The following tumors have been observed in connection with the meninges: fibromata, lipomata, angiomata, sarcomata, epitheliomata, papillomata, myxomata and melanotic sarcomata. Finally, dermoid cysts and odontomata occur in the brain or in its immediate neighborhood. In a case recorded by Roth in a goat the brain tissue was reduced to a layer a few millimeters thick owing to the presence of an odontoma weighing 310 g. From a clinical point of view tuberculosis of the meninges must also be included, this being of very common occurrence in cattle.

Tumors of the cranial bones may cause injury after breaking through the bones, by causing the development of exostoses on the inner surface of the cranial bones or they may develop directly on the inner surface of the cranium. Similar effects may be produced by tuberculous growths (Moussu and Frick) and by actinomycotic lesions (Pieroni). Finally, neoplasms in the neighborhood of the parotid may extend through the foramen lacerum into the cranium.

**Pathogenesis.** The injurious effects of tumors in the cranial cavity are due to the rise of intracranial pressure which they occasion. This varies with the size of the growth and at first affects its immediate neighborhood only, but owing to more extensive growth may involve more distant portions of the brain. In this way nerve cells and fibers are subjected to pressure and even destroyed. Many of the cells round about may be stimulated.

**Anatomical Changes.** In cases in which the tumor is in the brain tissue itself the portion containing it is enlarged, its convolutions flattened out and the medullary substance pale in color and dry. Tumors of this kind are generally found in the hemispheres, in the cerebellum and exceptionally in the corpus callosum (Cadéac), in the pituitary body (Wolff, Mollereau), in the olfactory bulbs (Marchand, Petit & Coquot) and in the pineal gland. Tumors in connection with the membranes or the bones are, as a rule, situated about the base of the brain. Tumors in connection with the base of the brain not only exert an effect on the brain tissue, but the roots of many of the cranial nerves may be also involved.

According to Zürn enlargement and induration of the pineal gland are met with in the fowl and the pigeon and cause cerebral disturbances.

**Symptoms.** Neoplasms that are by no means small may be found at the postmortem of animals which have shown no symp-

toms during life. Tumors in certain parts of the hemispheres, the ventricles and other parts of the brain may reach a considerable size without of necessity causing any symptoms. The reason of this probably is that owing to the slow development of the growth the brain accustoms itself to the altered conditions. In the cranial cavity of a horse that had shown no symptoms during life Blanc found a melanoma as large as a hen's egg.

The general cerebral symptoms presented are a more or less pronounced and slowly progressive dullness and awkwardness, which in some cases may increase until there is actual loss of intelligence. In the case described by Roth mentioned above the goat behaved as if it had no brain. Hand in hand with the dullness there is slowing of respiration and of the pulse, and peristalsis is retarded. Very often there are attacks of giddiness or forced movements, the animal walks in circles or leans up against the wall of the stable with the head drawn round to the side (Brade, Jessen). It is only exceptionally that severe symptoms develop within a short time and cause the death of the animal, but this is particularly the case in tuberculosis of the brain.

From time to time there may be symptoms of excitement and in cases of tuberculosis in which there are lesions involving the brain in cattle there are epileptiform seizures at increasingly short intervals as the disease progresses.

In other cases the muscular spasms are limited to the muscles of the head and neck or other parts of the body and in many cases only mystagmus and trembling are observed, and especially during movement.

Careful examination will reveal congestion of the optic papillæ as has been shown by observations of Scott and Wolff. Amaurosis has in some cases been observed in connection with these lesions, but in many cases the sight appears to be unaffected.

The local symptoms presented will, of course, depend upon the position of the tumor. As a rule, there is paralysis of certain nerves which gradually develops, and in the later stages may be severe, and may be extended to nervous tissue in the neighborhood. Hemiplegia, hemianesthesia, cerebellar ataxia, Jacksonian epilepsy, blindness are sometimes observed. In some cases, there are alterations in the shape of the cranium.

The following focal symptoms have been recorded in the cases which have been described with accuracy: Amaurosis of the left side owing to the presence of a tuberculous center the size of a hazelnut posterior to the decussation of the optic nerves (Uhlig). Muscular atrophy on the left side of the face with limited mobility and sensitiveness of the left half of the upper lip in a dog as the result of the presence of a glioma as large as a hazelnut which enclosed the Gasserian ganglion and extended backwards as far as the nuclei of the VI-VIIIth cranial nerves (Gratia). A similar case was observed by Lydtin in a horse, but the atrophy was limited to the muscles of mastication. Paralysis of the facial nerves on the right side with lateral displacement of the head and neck to the same side, and dragging of the left hind foot were observed in a horse having a sarcoma starting

in connection with the right petrous temporal bone and extending under the right half of the cerebellum, along the roots of the facial and auditory nerves as far as the pons and the restiform bodies on the right side (Pr. Mil. Vb., 1891). Paralysis of the facial nerves and complete blindness in cases in which except for large cholesteatomata\* in the lateral ventricles no (?) other lesions of the brain were found (Walley). Nystagmus, incoordination of movement, increased sensitiveness of the limbs (?) due to a tumor situated behind the cerebellum (Rutherford). Paralysis of the left half of the tongue and partial paralysis of the same side of the body owing to the presence of a sarcoma in the left half of the medulla oblongata immediately behind the decussation of the pyramids (Hallander). Twisting of the left eyeball in the outward direction was caused by a tumor originating in connection with the petrous temporal bone (Frick). Cadiot and Rogier observed cerebellar ataxia in a dog having a tumor in one-half of the cerebellum. Hébrant noticed unsteady gait, giddiness, swinging movements of the legs, exaggeration of the patellar reflexes, and in the later stages movement in circles to the right and right-sided facial paralysis in a dog having a sarcoma between the posterior vermis of the cerebellum and the right supero-lateral surface of the medulla oblongata. Cadéac and Roquet record paralysis of the trigeminal nerve in a dog having an endothelioma involving the root of that nerve. In a case recorded by Besnoit of tuberculosis of the brain with multiple centers the size of a nut and one larger area of softening in the right hemisphere there was blindness on the left side.

**Diagnosis.** A diagnosis can only be arrived at when in addition to symptoms of a general nature indicating pressure on the brain, there are local symptoms present. It must be borne in mind, however, that a more or less similar train of symptoms may be caused by animal parasites, abscesses, and in many cases by hydrocephalus internus and chronic meningitis. The differential diagnosis is not difficult when certain accessory circumstances, such as primary neoplasms in some other organ, tuberculosis, alteration of the shape of the cranium, suggest the presence or otherwise of a growth. The diagnosis of a tumor may, in all cases, be based upon the local symptoms, although not with absolute certainty, when these have gradually progressed without intermission, fever is absent, and there is congestion of the optic papillæ.

On the grounds of a careful examination of a case occurring in a foal Wolf gives the following symptoms of tumor growth in the region of the pituitary body: Depression, vomiting, congestion of the optic papillæ with resulting atrophy of the optic nerves, ptosis, gradually progressive loss of consciousness, retardation of the respirations and pulse. Further investigations must show whether motor and sensory disturbances are also present.

**Treatment.** In this connection no experiments have been made upon animals up to the present. No results are to be expected from internal treatment; at the most long-continued administration of potassium iodide or some other preparation of iodine might possibly be tried. Provided the diagnosis be practically certain and that it is supposed that the neoplasm is close to the roof of the cranium, surgical interference might be resorted to.

**Literature.** Besnoit, *Rev. Vét.*, 1906, 577 (Lit. on cerebral tuberculosis).—Bissange & Naudin, *Rec.*, 1904, 5.—Cadéac & Roquet, *J. Vét.*, 1908, 65.—Hamoir, *Ann.*, 1906, 232, 391.—Hébrant, *Ann.*, 1904, 438.—Marchand, Petit & Coquot, *Rec.*, 1906, 81.—Marchand, Petit & Pecar, *ibid.*, 1907, 25.—Peter, *B. t. W.*, 1898, 505.—Petit, *Bull.*, 1906, 85.—Roth, *Über eine intrakran. Dental-exostose usw.*, Diss. Zürich, 1888.—Schenk, *W. f. Tk.*, 1906, 705.—Schmey, *A. f. Tk.*, 1910, XXXVI, 121.—Wetzstein, *Studien üb. Tuberk. d. Zentr. Nervensystems*, Diss. Zürich, 1907 (Lit. on cerebral tuberculosis).—Wolff, *A. f. Tk.*, 1906, XXXII, 363 (Lit.).

\*See footnote, page 656.



### 13. *Gid.* *Coenurosis.*

(*Drehsucht, Drehkrankheit* [German], *Tournis* [French], *Gid, Staggers, Turnsick.*)

*Gid* is a chronic disease of ruminants, and especially of the sheep, and is caused by the *Cœnurus cerebralis*, the cystic stage of the *Tænia cœnurus*. In addition to other symptoms indicative of cerebral disturbance there are very frequently forced movements.

**Historical.** The *Cœnurus cerebralis* was first recognized as the cystic stage of a tapeworm by Leske in 1780, and the nature and etiology of the disease by Kuchenmeister in 1853. This was confirmed by other authors at a later date (Haubner, May, Gurlt, Gerlach, Leuckart, Baillet, Fürstenberg), and the *Cœnurus cerebralis* was recognized as the cystic stage of the *Tænia cœnurus*.



Fig. 91. *Cœnurus cerebralis*. Natural size.

**Occurrence.** The disease is known in all countries. The sheep is most commonly attacked, and the disease occurs more rarely in cattle and still more rarely in the goat. It is only exceptionally that the disease is met with in the horse, camel, dromedary, antelope, rabbit and hare. In wet seasons, the disease among sheep may amount almost to an epizootic. Trinchera observed outbreaks among calves, and Bauer among calves and adult animals.

**Etiology.** The *Coenurus cerebralis* is a single cyst which may be as large as a hen's egg and is filled with a colorless or pale yellow liquid. Through the delicate translucent wall one can distinguish small white specks, the

size of poppy seeds or smaller, each of which is an invaginated scolex. These are, as a rule, arranged in smaller or larger groups (fig. 91).

The cysts develop from embryos set free in the stomach from eggs of the *tænia*. Having bored their way through the wall of the stomach or intestine by means of their hooks, the embryos are apparently carried by the blood-stream to the cranial cavity of their new host, where they lose their hooks and become converted into small cysts. Exceptionally, they are carried into the vertebral canal. With further development the scolices are formed, thus producing the typical cysts.

An embryo in the central nervous system reaches the size of a pea in about 24 days, and scolices are first found about the 38th day. About three months are required for the full development of the cyst.

Views are somewhat divergent regarding the migrations of the embryos. One view is that the embryos provided with hooklets migrate into the connective tissue, probably around the blood vessels and enter the cranium by the foramen lacerum. This view is supported by the fact that after experimental infection minute tracks are found in the loose tissues of the thoracic and abdominal organs (Baillet) and also the fact that the first symptoms make their appearance after a week. As an explanation as to why the cysts principally develop on the upper surface of the hemispheres one must suppose that the embryos are able to develop more easily there because the furrows between the convolutions are deeper and the pressure is less. According to the other view, which is especially upheld by Möller, the escape of the embryos is by way of the blood stream. The occurrence of cerebral embolism due to worms, the absence of perforations or inflammatory changes at the base of the brain or in the dura mater support this view. The latter view is probably correct since there are other examples of the escape of worms from the intestine by way of the blood.

The power of resistance of the eggs of the tapeworm is apparently great, since eggs remain alive after the disintegration of the proglottides provided that the superficial layers of the ground or the grass are sufficiently moist. Gurlt saw a case of severe infestation after three weeks. The eggs do not resist desiccation for long and according to Röhl desiccation for two weeks in the open air kills them. Experience shows that infestation is rare in animals that are stall fed, because there is little opportunity for the contamination of food or drinking water with the feces of dogs under these circumstances. The resistance of the cysts is still less. According to Perroncito they are killed by raising the temperature to 38° to 41° C. and then cooling.

**Natural infection** is due to the ingestion of grass or water contaminated either with the proglottides passed out with the feces of a dog or with eggs of the *Tænia cœnurus* after the disintegration of the proglottides. The disease attacks sheep and other herbivora. The shell of the egg is dissolved by the gastric juice, and the liberated embryo commences its migration towards the central nervous system. The tapeworm develops principally in the intestine of sheep dogs, owing to the culpable habit of shepherds of giving the brains of diseased sheep to the dogs, and the carcasses of sheep that have not been buried properly are also dug up by the dogs.

The occurrence of the disease is naturally bound up with the number of dogs, but the state of the weather, especially in rainy springs and autumns, is also an important factor, because the dampness favors the vitality of the embryos. Infection through contaminated water may take place throughout the year (Moussu). According to Diem, experience shows that the disease occurs more frequently among the animals of farmers whose meadows are close to the road where dogs can deposit their feces.

**Susceptibility.** The sheep is the most susceptible of all, cattle and other ruminants being more rarely affected. The horse and many wild herbivora are only slightly susceptible. The disease generally occurs in young animals, the sheep being affected up to one year old and cattle up to two years. Even experimental infection fails in older animals save in exceptional cases. The ox is an exception in that infection is not abso-

lutely rare in older animals. The increasing resistance offered to the disease with advancing age in all probability is connected with the greater delicacy of the tissues in young animals, this favoring the migrations of the parasites.

According to the older authors *cœnurus* cysts are exceptionally found in lambs at the time of birth or a few days after (Simonds, Hering). If these observations are correct the embryos must have entered the body of the fetus by way of the placental blood vessels.

**Pathogenesis.** The oncospheres reach the cortex by way of the blood vessels of the pia mater and may penetrate into the medullary substance. During their passage they make tracks, and set up inflammation in the immediate neighborhood. This circumscribed inflammation causes no disturbance of the functions of the brain, provided the inflamed areas are few in number; but where the parasites are numerous, there may be severe disturbances, owing to the larger number of small centers. The development of the embryos into cysts increases the intracranial pressure in proportion to the size of the cysts, this pressure causing atrophy of the nervous tissue in the neighborhood and in more remote parts of the brain. As a result of this destruction of nervous tissue, both general and local symptoms make their appearance.

**Anatomical Changes.** In the sheep in the acute stages one can see small tracks in the cortex of the upper portion of the hemispheres corresponding with yellowish purulent streaks in the arachnoid, while the inner surface of the dura may be covered with a purulent layer. One can also see green or greenish-yellow bladders containing pus-like material contained within translucent membranes. These vary in size from a pin's head to a linseed and they are surrounded by a layer composed of the remains of destroyed tissue and calcified granules, while there may be small hemorrhages round about. The fluid contained in the ventricles may be increased in amount and turbid, especially if, as is exceptionally the case, the small cysts have invaded the venous plexuses.

In the chronic stage, as a rule, only one or two and more rarely as many as six cysts may be found. Hink records a case in which there were 17 and Huzard even 30. Sometimes small granules, the remains of dead embryos, can be recognized in the membranes. The larger cysts are generally found on the convex surface of the brain or in the hemisphere, but they may be occasionally found in connection with the cerebellum, or the base of the brain. Their connection with the pia mater is generally easily discovered. The brain tissue appears atrophied and anemic, and in the immediate neighborhood of the cysts is converted into a reddish granular mass, or in some cases it may have a viscid consistency. In the great majority of cases one, and more rarely both, the hemispheres are de-



stroyed, and the basal ganglia flattened. In some cases, one hemisphere may be entirely replaced by a cyst. Exceptionally, the hydatid is quite free in the excess of fluid in the lateral ventricle (Hering, Hutyra & Marek) and may pass from there through the foramen of Monro into the ventricle of the opposite side or into the third ventricle.

Cysts on the surface of the hemispheres may cause the destruction of the roof of the cranium immediately over them (two cases Hutyra & Marek), and even of other portions, generally the frontal or parietal bones. In such cases, the bone becomes as thin as paper and quite translucent, or the bone tissue may quite disappear over a circular area, leaving the cyst covered only by the membranes of the brain, the periosteum, and the soft tissues over the cranium.

Out of 100 cases Kolb found the cyst in 32 cases on the left side, 68 on the right, and in 5% of cases in the cerebellum.

In rare cases, the cysts develop in the vertebral canal (see compression of the spinal cord).

In **cattle** the cysts sometimes attain the size of an orange, and there may be a number present (Vollrath found twenty-three, and Pfab sixty). They may be the cause of exactly similar lesions of the brain and also of atrophy of the roof of the cranium. If they are situated under the frontal bone in calves they may perforate the still single layer of bone; but in older animals, the inner layer is forced outwards and the frontal sinus is completely obliterated. The outer wall may also appear to bulge outwards (Greve), or may even be atrophied over a circular area (Pfab).

In the **horse**, as a rule, only single cysts are found, either on the upper surface of one of the hemispheres, or in a lateral ventricle. They have been found in one case between the hemispheres and the cerebellum, and in one case in both hemispheres.

In animals that have been ill for some time, symptoms of anemia and cachexia are generally seen. Sometimes, one sees in the thoracic or abdominal wall, or in the loose connective tissue small rounded greenish-yellow nodules about the size of a pea. On section these are found to contain a pulp-like mass enclosed in a membranous envelope which in all probability represents the remains of dead embryos (Röll, Neumann). Exceptionally well-developed hydratids may be found in the subcutaneous connective tissue (Nathusius, Eichler), in the thyroid and in the muscles (Rabe).

**Symptoms.** In the **sheep** two stages separated by a long interval are recognizable during the course of the disease. The first stage is cerebro-meningitis, set up by the migrations of

ity. It may be anything from two to six or eight to ten days, after which the symptoms, having reached a certain stage, gradually disappear, and the animals apparently recover.

The second stage is preceded by a latent period of three to six months, and as a rule in flocks of sheep no symptoms are observed during this period. Now and then, the disturbances of the functions of the brain are indicated by a certain dullness, especially when the weather is warm or in damp sheds. After the latent period the symptoms which are characteristic of the second stage, gradually develop.

Disturbances of consciousness are prominent among the general brain symptoms. The animals remain separated from the flock, move with unsteady gait, the feet being lifted high. They stumble along with the head depressed or held high. As a rule, the head is turned around to one side (see figs. 94 and 95) and the animals while standing still, allow the head to drop, or they stand with the forehead pressed against the wall (fig. 93). There is a vacant expression and the appetite is variable. Finally the giddiness, which results in a stumbling gait, must be referred to disturbances of consciousness.

In many cases there are convulsions. Individual groups of muscles may be involved, for example, the muscles of mastication, causing the animal to grind its teeth and the saliva to froth.



Fig. 93. Gid.

In other cases, there may be epileptiform seizures. Forced movements may be produced by the disturbances of consciousness. These, however, are not constant in nature, and consequently are of no value for determining the situation of the cyst. The types of movement vary from case to case. There may be movements in circles, rotation at one place, stumbling, falling on one side, forwards or backwards.

The veins of the optic disc and the surrounding tissue appear very congested, the arteries on the other hand are constricted. There is sometimes edema of the optic nerves and a diffuse cloudiness of the optic disc indicating neuro-retinitis (Bouchut).

Among the focal symptoms there is sometimes observed squinting, resulting from compression of the nerves of the muscles of the eyes, and there are also forced movements which are constant.

Animals sometimes move in circles, first in one direction, and then in the other. This, according to Diem, is seen particularly early in Simmenthal cattle. As the disturbances of consciousness become more marked, there is developed a condition resembling staggers. This stage is especially marked in young animals in which the symptoms are striking, but in older animals, it often escapes observation.

The second stage sets in in from three to six months (Pfaff) but it is not sharply marked off. The symptoms presented closely resemble those observed in the sheep. Many affected animals cannot find their places in the shed, especially if this lies in the opposite direction to that in which they tend to move



Fig. 95. Lateral flexion of the head in gid.

when at pasture (Cruzel). It is very difficult to turn affected animals or move them backwards; they cannot see objects in their way and fall over them (G. Lövy). In one case recorded by Trinchera, in which there was a hydatid in the neighborhood of the corpora quadrigemina on the right side as large as a hen's egg, there was in addition to movements in circles, partial paralysis and atrophy of the limbs of that side.

Eder is inclined to refer the paralysis of the fore-limb seen in a calf affected with staggers to the presence of the cyst in the brain. Simultaneous with the movement in circles, there is a tendency to hold that side of the head which is turned



towards the interior of the circle lower than the other side. Not rarely and especially in the later stages of the disease there is extension of the head (Albrecht).

The frontal and parietal regions, and the roots of the horns feel warmer than normal and exceptionally the frontal region may bulge forward (Greve). In animals up to one to one and one half years old, percussion of the cranium, provided the cyst is superficially placed, gives an unusually hollow sound, similar to that obtained by percussion, immediately below the horn core (Albrecht, Merkt). At the same spot the resistance is elastic. Now and then the roof of the cranium is yielding (Albrecht), and in many cases the use of the percussion hammer produces symptoms of pain.

For percussion of the cranium Albrecht uses a light hammer, but Diem prefers one that is fairly heavy and has one end round and the other square. Pfab has recently constructed a useful hammer for this purpose. Percussion hammers of the usual type are useful for the discovery of quite superficial cysts. The whole of the frontal region should be percussed with the head extended after the hair has been clipped off.

As the appetite falls off there is gradual emaciation, and if the animal be not slaughtered in time, it finally gets into a condition of complete cachexia.

In the isolated cases that have been observed in horses, the functional disturbances have been somewhat severe. In a case recorded by Schwanefeld, there were symptoms of acute cerebro-meningitis (the cyst being on the upper surface of the hemisphere). Frenzel observed emaciation and dilatation of the pupils, and from time to time, circular movements. In Gotteswinter's case, the horse which was previously quiet, became excitable and vicious, and later showed symptoms of sleepy staggers. In Zündel's case the disease which lasted four months, started with amaurosis. Later there were attacks of giddiness, while the horse was at work, the head being held to the left and the animal going in the same direction. It was found to be impossible to turn it the other way. The cyst was found in the right hemisphere under the frontal lobe. In addition to these symptoms, rotation, backing and sleepy staggers have been observed,

**Course.** Apart from those cases in which the animals die showing symptoms of acute encephalitis at the time of infection, the disease tends to be chronic and last for months. In a proportion of cases, symptoms make their appearance ten to fourteen days after infection, the condition lasting sometimes for a week. These symptoms then disappear, and the characteristic symptoms set in after three to six months. In the majority of cases, the disease tends to begin with symptoms indicative of pressure on the brain. The length of the interval between infection and the appearance of the characteristic symptoms depends upon the severity of the infestation, but the situation of the cysts is not without some influence on this. The subsequent course of the disease depends upon the same factors. There are cases in which the symptoms develop rapidly and

increase in severity so quickly that the animal dies in the course of a few days. This stage, as a rule, lasts from two to six weeks, but it may extend, and especially in cattle, to two to three months, or even longer. Gières records a case in which it was eight months; and Pfab, one in which it was more than a year.

Death is generally due to weakness and anemia, but it is sometimes quite sudden and resembles apoplexy.

**Diagnosis.** During the acute stage the symptoms are those of acute cerebro-meningitis, and during the chronic stage those of increased intracranial pressure. An opinion as to the cause of the increased pressure may be based upon the local symptoms, the examination of the roof of the cranium, and the further course of the disease, provided the preliminary enquiry affords exact information. In the absence of such information, diagnosis passes beyond the bounds of possibility, although in practice the disease is generally easily recognized, and especially in the sheep, on account of its frequent occurrence and still more from the postmortem examination of some of the animals.

In connection with differential diagnosis, sunstroke and acute meningitis must be taken into consideration. Apart from the sudden onset of the disease, these cause severe depression from the outset. Forced movements and particularly movements in circles are only rarely observed. There is congestion of the mucous membranes of the head, and the course is very acute. When the disease occurs enzootically among sheep, it may be confounded with infectious cerebro-spinal meningitis, especially when housed animals are attacked. Apart from the diagnostic symptoms of meningitis, a diagnosis may be always based upon a postmortem examination. Severe cerebral symptoms may be caused in sheep by the larvæ of the *Cæstrus ovis*. In such cases, there are only symptoms of acute inflammatory disease of the brain and its membranes without forced movements, and besides valuable information is furnished by the catarrh of the nasal cavities and sinuses which is present at the same time. One must not lose sight of the fact that both diseases may exist at the same time. Errors should scarcely be made in other cases of catarrh of the nose and sinuses.

Tumors of the brain, tuberculosis of the brain, or its membranes in cattle and in particular echinococcus cysts may set up similar symptoms.

In such cases the possible presence of similar tumors, tuberculosis, or parasites in other organs, affords a certain amount of information, although this does not enable one to make a certain diagnosis. With regard to tuberculosis, the presence of lesions in the eye is decisive. For the rest, attenuation of the roof of the cranium has not up to the present been observed in any other disease of the brain. In the horse

chronic dropsy of the ventricles must be considered, but in this case differential diagnosis may be based on the absence in cœnurosis of the acute attacks that not rarely occur in internal hydrocephalus. There is a possibility of confusion with epilepsy in those cases in which occasional attacks of general spasms occur, but in epilepsy there is complete absence of any evidence of brain trouble in the intervals between the attacks.

A local diagnosis is only possible when there are pronounced localized symptoms (movements in circles always in the same direction, cerebellar ataxia, rolling, functional disturbances of certain cranial nerves, Jacksonian epilepsy), and there is compressibility of the roof of the cranium at one particular spot, or percussion yields a hollow sound, but as mentioned above, the spot does not always mark the seat of the cyst.

The connection between the nature and direction of the forced movements and the situation of the cyst, is seldom so intimate that the situation of the cyst can be determined without further investigations. This is true, both for the cases in which there is a single cyst, and in cases in which there are large numbers. Comparison of large numbers of results gives the following points as being common to the minority of cases. In most cases, the cyst is in that half of the brain towards which the forced movements are made. In cases in which the animals move in circles, the cyst is situated in that half of the brain which is turned towards the center of the circle and is situated close to the roof of the cranium. Rotation about a fixed point indicates the presence of a cyst in the depth of the opposite hemisphere, or in the floor of the lateral ventricle, in which case the corpora quadrigemina are subjected to pressure. In some cases movement in circles towards the sound side is seen. When animals force their way forwards with the head depressed, the cyst is as a rule, in the frontal lobes directly in front of the corpus striatum; while in cases in which there is rolling, it is situated at the base of the cerebellum or on the pons. If the cyst be in one of the hemispheres of the cerebellum, the vermiform process, or the occipital lobe, there is evidence of great giddiness and a staggering gait. In cases in which the head is drawn forcibly backwards, the cyst is usually to be found in the neighborhood of the tentorium cerebelli between the cerebrum and cerebellum.

In 76 animals that were trepanned or examined postmortem Scholtz found the cyst in 17 cases on that side of the brain that was turned towards the center of the circle.

**Prognosis.** The disease may terminate at the stage of excitement, owing to the death of the embryos (about two per cent of cases, Zürn), but prognosis is unfavorable, even in the early stages. In the second stage, prognosis may be made more or less favorable by surgical interference. Results obtained up to the present show that provided the operation be done in time about a third of the number of sheep, and in cattle under favorable conditions, not rarely a half or even two thirds of the animals, may be saved.



**Treatment.** In the first stage cold applications to the head and purgatives are indicated to alleviate the symptoms produced by the inflammation.

In the second stage only removal of the cyst by trepanning can lead to recovery.

Under the following circumstances surgical interference is indicated: a superficial and accurately localized situation of the cyst, the appetite maintained to some extent, and the animal in satisfactory condition, the absence of spontaneous convulsions, and finally, when slaughter is not desirable. In connection with the operation in cattle, the breed of the animals must be taken into consideration. Results obtained by a number of Bavarian veterinarians (Imminger, Braun, Diem) show that trepanning is attended with great difficulty in the case of Simmenthal cattle on account of the extraordinary thickness of the cranial bones, the tough structure of the cysts; and finally, the special susceptibility of the animals to the operation. Diem's results (87% of cures) indicate that, provided certain precautions are taken (ligation of blood vessels, the cyst be not probed about or infected, the wound be thoroughly cleansed and washed out after operation) as good results may be obtained with this breed as with others. The species of the diseased animals should also be taken into consideration. Sheep are of less value than cattle, and not infrequently they harbor a large number of cysts, points which are opposed to the operation being undertaken, and to its success. In cases in which the symptoms indicate the presence of cysts, but their exact situation cannot be determined, it is advisable to wait for two to eight weeks in the case of cattle so that the chance of localizing the parasite may be increased. According to Diem, pregnancy is no bar to the operation.

In operating, the entire cyst should be removed, the results following removal of the contents only being less favorable. As a general rule, it appears to be advisable to remove the liquid slowly and with pauses as a sudden alteration of intracranial pressure may cause fatal paralysis or general convulsions and hemorrhage.

The seat of operation depends upon the case and especially upon the local lesions presented by the cranial wall (see pages 666 and 667). If such indications be absent the operation should be undertaken only at the special desire of the owner. With experience the cyst will in most cases be found, and especially if the animals have shown symptom of moving in circles or forwards. In horned breeds of sheep the middle portion of the occipital lobe lies 12 mm. in rams and 16 to 20 mm. in ewes behind the middle point of the line joining the horn cores, and the frontal lobes immediately behind the inner portion of the horn or the horn core. In hornless breeds the following are the guiding points: The area included between lines joining the bases of the ears and the eyes and lines joining the base of each ear with the eye on the same side is divided medially by a vertical line, and the operation is carried out at the mid-point of the areas so marked out. In cattle the proper seat of the operation is at a point 4 cm. above a line joining the brows of the eyes and 1.5-2 cm. from the middle line (Albrecht). In any case the middle line must be avoided so as to escape injuring the longitudinal sinus. More exact directions will be found in text books on surgery, and references to the operation in cattle will be found in the publications of Merkt, Albrecht, Diem and Pfab.

In cases in which the operation is successful and recovery is going to take place, there is a striking improvement in the

animal's condition within the next few days; the depression disappears; there are no forced movements, the appetite returns and in a week or two, the animal appears to have recovered. Cattle as a rule recover more slowly. For the first few days after the operation, they are so weak that they have to be fed artificially. Recovery generally takes two or three weeks, but there are many exceptions to this. If, after the operation, symptoms make a fresh appearance and are associated with fever, encephalitis or meningitis must be suspected, and the animal slaughtered as soon as possible. The same holds good for those cases in which symptoms appear after a longer interval, because it is obvious that in such cases one or more cysts remain behind in the cranium. Now and then, a second operation leads to a definite recovery (Renner).

The proportion of recoveries following operation is of course very variable. In sheep Scholtz records a percentage of 25, Demann 33%, Kuhlmann 25 to 35%, while in cattle Diem had 91% of recoveries in 24 animals operated upon and Pfab 59% of 58 cases. Half of Merkt's cases and all of Renner's recovered. These good results are quite the exception, accidents being recorded in the majority of cases (Vollrath, Braun).

The only other method of treatment that will be mentioned here is that advised by Hartenstein and used by Nocard with good results in two cases. The sheep is enclosed in a box with its head extended through a hole of suitable size and, with the exception of feeding times, the head is subjected to a stream of cold water which is supplied through a rubber tube from a vessel fixed at a height. The irrigation must be continued for three days and then repeated at longer intervals until all symptoms disappear. Postmortem examinations have shown that under such treatment the cysts die. De Mia effected cures in two calves up to a year old by applying ice compresses to the head for 12 days.

**Prophylaxis.** Efforts must be made to protect sheep and cattle from infection from dogs. In this connection a reduction in the number of sheep dogs is indicated, and tapeworms should be expelled from the dogs every two to three months. During treatment the dogs must be isolated and their feces burnt or buried deeply. Care must also be exercised that the dogs are afforded no opportunity to get hydatids from animals that have died or have been slaughtered. This is best accomplished by burning or cooking the affected brains. If certain fields are known by experience to be dangerous, the young cattle which are more susceptible, should be kept off them, especially during wet weather.

**Literature.** Albrecht, Monh., 1894, V, 337.—Bauer, W. f. Tk., 1901, 15.—Braun, *ibid.*, 1906, 441.—Diem, *ibid.*, 1906, 881.—Gotteswinter, *ibid.*, 1894, 378.—Greve, Mag., 1835, 23.—Hering, Rep., 1855, 20; 1859, 247.—Kunz, Schw. A., 1893, XXXV, 62.—Leblanc & Fréger, J. Vét., 1907, 193.—Lövy, Vet., 1895, 222.—De Mia, N. Ere., 1904, 109.—Möller, D. Z. f. Tm., 1875, I, 425.—Pfab, Münch. t. W., 1910, 73 (Lit.).—Renner, W. f. Tk., 1878, 174.—Schmid, *ibid.*, 1906, 706.—Scholtz, Pr. Mt., 1869-70, 147.—Schwanefeldt, A. f. Tk., 1885, XI, 230.—Trinchera, Clin. Vet., 1893, 485.—Vollrath, W. f. Tk., 1905, 791.—Zimmermann, Vet., 1901, 689.—Zürn, Tier. Parasiten, 1882, 139.

**Other Parasites of the Brain.** In the pig, and more rarely in the dog, the *Cysticercus cellulosæ* develops in the brain, and the *Cysticercus bovis* has been found in calves by Deleidi & Reggani. Lesbre records

the occurrence of the *Cysticercus pisiformis* in the brain of a dog.—**Echinococcus cysts** have now and then been found in the brain of the horse (Oeltjen, Boschetti, Dollar, Gützloff, Glocke), and in cattle (de Angelis, Reali, László). The affected animals either showed simply symptoms of dullness, or there were attacks of mania; the head was held obliquely, and percussion of the cranium yielded a hollow sound (Reali).—The **larvae of sclerostomes** sometimes occur in the brain of the horse, and may give rise to an acute hemorrhagic meningo-encephalitis, and possibly also to dilatation of some of the arteries of the brain (Abilgaard, Le Bihan, Albrecht, van Heill). The occurrence of the **larvae of *Gastrophilus*** has been recorded (Dieckerhoff, Lindström, Siedamgrotzky, Johné). The penetration in all probability took place via the Eustachian tube or from the guttural pouches through the foramen lacerum. As a rule, they remain in the base of the brain and cause an acute rapidly fatal meningo-encephalitis. On a few occasions the **larvae of the *Hypoderma bovis*** has been found in the horse (Boas, Poulsen, Segelberg, Railliet), and on one occasion in Hungary, in the ox. The animals showed symptoms of a rapid and acute meningo-encephalitis. Widakowich found a sexually mature roundworm in the enlarged pituitary body in a cat, and Lafargue in a horse showing symptoms of meningitis two amphistomes in a subarachnoid hemorrhage.

Apart from cysticercosis, no case has been recorded of parasites causing functional nervous disturbances during life.

**Literature.** De Angelis, N. Erc., 1903, 62.—Lafargue, Rec. d'Hyg. et de Méd. Vét. Mil., 1909, X, 705.—Martin, Rev. Vét., 1907, 741.—Railliet & Ducasse, Rec., 1901, 207.—Reali, Clin. Vet., 1900, 259.—Siedamgrotzky, S. B., 1884, 15.—Widakowich, Cbl. f. Bakt., 1905, XXXVIII, 447.

#### 14. Bulbar Paralysis. Paralysis bulbaris.

##### (*Paralysis glosso-labio-laryngea.*)

Bulbar paralysis is the term used to indicate combined paralysis of the cranial nerves arising from the floor of the fourth ventricle (sixth to twelfth pairs). The disease may be uncomplicated, or it may accompany other diseases of the brain (meningitis, encephalitis, hemorrhage, contusion).

##### (a) Acute Bulbar Paralysis.

Paralysis of the nerves arising from the medulla oblongata may be caused by hemorrhage that is limited to the medulla, plugging of the vessels, or inflammation. In this connection, encephalitis comes into consideration first. In many cases of encephalitis due to distemper, and in the majority of cases due to rabies, the disease sets in with bulbar paralysis. So far, no anatomical reason has been found for the cases of infectious acute bulbar paralysis observed in the cat and dog in Hungary (see Vol. I), although Marek records a perivascular infiltration in cattle that have died of the disease. Finally, poisoning by bunt and rust in some cases causes symptoms of bulbar paraly-



sis (see page 215). All these forms have been dealt with in the chapters devoted to the particular subjects.

There appear to be other diseases than those mentioned above that occur in animals, and are associated with bulbar paralysis. Stietenroth observed a bulbar paralysis terminating fatally in seven to twelve days in three horses, the exact nature of which was not determined. Thomassen mentions the occurrence in Belgium and Holland of a contagious disease among young horses associated with suddenly occurring difficulty of masticating and swallowing, and paresis of the limbs. Histological examinations revealed no alterations save that there was a reduction in the number of ganglion cells in the neighborhood of the nuclei of the hypoglossal, glosso-pharyngeal and vago-accessorius nerves and there was chromatolysis, vacuolization and marginal disposition of the nuclei. Thomassen identifies the disease with the progressive bulbar paralysis observed in Belgium, but owing to essential differences in the course of the disease, it should not be considered as the same. From time to time, cases are recorded in which paralysis of the nerves of the bulb is an outstanding feature. Little definite information can at the present moment be given with regard to these and similar diseased conditions. The principal conditions included under the term are encephalo-myelitis of various types, diseases of the peripheral nerves, and poisoning.

**Literature.** Dexler, *Ergebn. d. Path.*, 1896, III, 507 (Lit.).—Döderlein, *W. f. Tk.*, 1905, 83.—Stietenroth, *B. t. W.*, 1899, 265.—Thomassen, *Monh.*, 1903, XIV, 1.

#### (b) Progressive Bulbar Paralysis.

**Occurrence.** There are observations to show that progressive bulbar paralysis occurs in the horse as an independent condition (Lagrange, Stockfleth, Gérard, Degive, Cadéac, Thomassen, Fröhner). The disease appears to have a tendency to break out sporadically every year in the northern parts of Belgium. Isolated cases have been recorded by Cadéac in France, Fröhner and Rosenfeld in Germany.

**Etiology.** Belgian authors and Thomassen are inclined to think that the cause of the disease is an intoxication due to white beets, there being no evidence that it is due to an infection. It is still unknown whether the disease actually involves the medulla oblongata or the nerves originating from it.

**Symptoms.** Clinically, the disease is characterized by a gradually progressing bilateral paralysis of the bulbar nerves. There is difficulty in swallowing in every case. The animal takes food greedily, chews it for a long time, but swallows only a part or none at all, the food returning partly through the nose

and partly through the mouth. Illumination of the interior of the pharynx may show it adhering to the posterior wall of the cavity. If there is simultaneous paralysis of the esophagus food may remain here. Drinking appears to be little disturbed at the outset. The secretion of saliva is increased. Paralysis of the larynx causes whistling or roaring and finally aphonia. As a result of paralysis of the tongue, swallowing is rendered still more difficult and the food collects in the mouth. This greatly interferes with drinking, and in many cases the horses try to get water into the upper part of the esophagus by suddenly lifting the head after having taken water into the mouth in a manner resembling that seen in the goose. The tongue undergoes a process of atrophy and its upper surface appears wrinkled. Evidence of paralysis of the nerves of the face soon makes its appearance and, owing to paralysis of the lips, food is prehended with the incisor teeth. Finally, there is paralysis of the muscles of mastication. In exceptional cases the paralysis is more pronounced and more extensive on one side than on the other. In a case of bulbar paralysis observed by Fröhner there was pronounced atrophy of the muscles of the back and croup.

**Course.** The disease lasts for five or six months or more, the animals becoming very emaciated owing to difficulty of swallowing. Debility or pneumonia due to the passage of food down the esophagus finally bring the disease to a fatal termination.

**Diagnosis.** Diagnosis is based upon the exclusion of abscesses in the pharynx, neoplasms, impaction with foreign bodies and diseases of the esophagus.

**Treatment.** The administration of thin pultaceous foods is indicated throughout the course of the disease, a stomach tube being used if necessary (see page 123). If the disease is not checked it appears advisable to consider the value of the animal as early as possible.

**Literature.** Cadéac, J. Vét., 1902, 519.—Degive, Ann., 1883, 12.—Fröhner, Monh., 1905, XVI, 550.—Rosenfeld, Z. f. Vk., 1905, 26.

## SECTION II.

### DISEASES OF THE SPINAL CORD.

#### 1. Acute Inflammation of the Membranes of the Spinal Cord. *Meningitis spinalis acuta.*

Under this term are included acute inflammations of the membranes of the spinal cord which commence either in the dura mater, the epidural tissues or the pia mater, and as a rule, spread to the other membranes and even the spinal cord itself, causing the production of a serous, sero-fibrinous, or suppurative inflammatory exudate.

**Occurrence.** Apart from cerebro-meningitis, the disease is of rare occurrence in the domesticated animals, but among them the majority of cases occur in the horse.

**Etiology.** Inflammatory conditions of the membranes of the spinal cord are always due to an infection. Now and then, cases occur as a result of influenza, strangles, pyemia, or septicemia. Hess records a case of purulent meningitis of the cauda equina in an ox that was associated with purulent cerebral meningitis. Apart from some calcified tubercles in the mediastinal gland and worm nodules in the small intestine, there were no lesions in the internal organs. Injuries such as blows on the vertebral column, docking the tail too short, are sometimes causally connected with the disease, in that they allow of the access of bacteria. Finally, in England, there is a contagious disease of the sheep characterized by a pyemic inflammation of the membranes of the cord which is known as "loping ill" (McFadyean).

By Fröhner, Cadéac and others distemper is thought to be causally related to an acute and clinically recognizable spinal meningitis, because in this disease there is an accumulation of a large quantity of clear liquid in the inter-meningeal spaces (hydorrhachis). The authors have never observed any clinical evidence of spinal meningitis in the many hundreds of cases of distemper that have come under their notice.

Not rarely the inflammatory processes spread to the membranes from surrounding tissues. This is especially the case in caries of a vertebra, the rupture of an abscess in the neigh-



borhood of the vertebral canal (Decosse, Ohm, Fröhner), or in cases of suppurative inflammation in the mediastinum or neck. According to Trasbot, inflammation may extend along the course of nerves from nerve plexuses to the membranes of the cord. This has been proved by Homén and Laitneu by the injection of streptococci into the sciatic nerve; the meningitis observed in one case of dourine may have been caused in this way. The circumscribed spinal meningitis which often develops is not of much clinical importance as it is obscured by the myelitis which develops at an earlier stage.

**Pathogenesis.** The nerve terminals in the membranes, the cord, and the nerve roots passing through them are irritated by the hyperemic dilatations of the vessels and the serous-celled infiltration which soon follows. In the later stages the conductivity of many of the nerve roots is lessened or even destroyed. In addition to this, there is an extension of the inflammatory processes from the membranes to the superficial layers of the cord itself. Intermixing of the toxic material with the cerebro-spinal fluid, generally causes the extension of the inflammation over a large area.

**Anatomical Changes.** In addition to the marked congestion of the vessels there is an exudate in the intermeningeal space which is turbid or even mixed with flocculi of fibrin. The vessels of the membranes may be surrounded by layers of fibrin of variable thickness, producing an appearance of streaks of pus. In many cases, the exudate is actually purulent. Not rarely the fibrinous or purulent exudate involves the nerve roots connected with the spinal cord.

The presence of a clear serous fluid between the membranes is in itself no proof of the existence of spinal meningitis. In animals in an advanced stage of emaciation there are often large quantities of clear fluid in the subdural space, but this is accounted for by the decreased resistance owing to the disappearance of the epidural fat.

**Symptoms.** The first symptom is increased sensibility of those parts of the body, the nerve trunks of which pass through the diseased membranes. As a result of this, the gait is particularly cramped and cautious, and the animals hold the vertebral column as motionless as possible and stiff. The sensibility of the skin (hyperalgesia) is further shown by the symptoms of pain exhibited if the skin be pinched or rubbed with the flat of the hand, especially in the direction opposed to the lay of the hair, or if a sponge soaked in warm water be placed in contact with the skin. The back is arched, and the animals become very restless, snap at the hand, and utter cries of pain. Similar symptoms are exhibited if pressure be exerted upon the muscles. In some cases, accurate investigation shows that this increased sensibility is restricted to certain parts of the body, a quarter, one half or a third of the body

(Spinola and Trasbot). In all probability, animals suffer pain without any interference from without, in that they, and especially dogs, often utter cries and moans, and persistently rub some part of the body and even bite it until the blood comes. The painfulness of the vertebral column is shown by percussing the spinous processes and during passive movements.

At this stage there are momentary spasms, fibrillar twitchings or cramplike contractions of the muscles. These contractions occur principally in response to external influences, and in some cases in response to the lightest contact with the skin, but in others as a result of heavier blows, pressure, or attempts to move the animal. In one case observed by Fröhner this caused a horse to rear. If the contractions of the muscles are persistent and involve the muscles of the back, the back is arched and the muscles are as hard as wood. Spasms of this kind frequently occur in the muscles of the limbs at the same time, and if the inflammation has extended to near the medulla oblongata the neck muscles may be thrown into spasm. Should the contractions involve the muscles of respiration and the abdominal muscles, respiration becomes hurried and superficial, the abdomen is tucked up; and there is retention of feces and urine, probably owing to contraction of the sphincters of the anus and bladder. In other cases, urine is passed frequently, the passage being associated with pain. Now and then, priapismus is observed in male animals. In the early stages, both kinds of reflex are exaggerated in the hyperalgesic area.

After the motor nerves have been deprived of part of their conductivity, paralysis of individual muscles or groups of muscles in the hyperalgesic area is observed, associated with reduction or complete loss of the previously exaggerated reflex irritability. At the same time, or more usually at a later stage, there is a reduction, but rarely a complete loss of sensibility in those parts of the body that were previously painful. These symptoms, as a rule, remain confined to a small area so long as the spinal cord appears to be uninjured.

Extension of the inflammation to the cord or compression of the cord by the exudate is rapidly followed by extensive and progressive paralysis and anesthesia of all portions of the body posterior to the most anterior limit of the area involved in the inflammation; in a word, there are symptoms of inflammation of the spinal cord. As a result of the paralysis, the gait becomes uncertain and in a short time the animal can no longer stand, bedsores very promptly appearing on prominent parts of the body, especially in large animals.

There is frequently elevation of temperature, and in many cases the onset of the disease is marked by high fever.

Exceptionally the course of the disease may differ from that described. There may be loss of sensibility from the outset (Hutyra & Marek) or there may be paralysis at once (Fröhner), and especially if the cord is affected from the commencement.

**Course.** The disease, as a rule, terminates fatally within a week, death being due in most cases to disease of the spinal cord. There are cases in which circumscribed inflammation of the meninges may persist for months.

**Diagnosis.** There is no special difficulty in making a diagnosis when the symptoms can be observed to make their appearance in the order given, but systematic observation is essential. Symptoms that are of particular value are the gradual disappearance of the irritability of the nerves, which is present in the early stages, until there is complete paralysis, and the forward extension of the inflammatory processes. With reference to the course taken by this disease, injuries to and compression of the cord are easily excluded.—Pachymeningitis ossificans may be distinguished by the chronic course and the slightness of the initial symptoms.—Acute muscular rheumatism may be differentiated by the painfulness of the muscles, while the sensibility of the skin remains normal, and the normal condition of the reflexes, tetanus by the trismus, and the absence of sensory disturbances; acute inflammation of the brain and its membranes by the pronounced dullness and the paralysis of the cranial nerves.—Polyneuritis sometimes occurs in animals, but in this condition there is exaggerated sensibility, but there are no muscular spasms.—In many cases, the differentiation of transverse or diffuse inflammation of the cord may be associated with difficulty. The chief distinction is that in spinal meningitis there is severe pain and muscular spasms over large areas. In inflammation of the cord there is at an early stage extensive sensory and motor paralysis, and disturbance of defecation and micturition are more frequently observed. Finally, experience shows that in cases in which the disease is confined to the cord there are no objective symptoms of pain.

**Treatment.** The increased excitability and reflex irritability seen in the early stages make it essential that the animal be placed in a quiet, clean box and provided with a soft bed, because bedsores are easily produced in cases of this kind. The affected parts of the body should be injected or rubbed several times daily with alcohol containing camphor or turpentine, and parts that are already partly paralyzed must be kept clean. Large animals should be placed in slings, should the increased irritability permit of it, and paraplegia has not become complete. Should the animal go down, the parts must be washed several times a day with a disinfectant and sprinkled with a disinfecting powder.

Cold applications should be used to oppose the inflammatory processes, and this is particularly the case in small animals in which the thin layer of muscles does not offer much opposition to the effect of the cold on the vertebral canal. India rubber



bottles of suitable shape, which should be filled with ice or with cold water which is changed at frequent intervals during the day, may be laid along the course of the vertebral column. Thick layers of tow may be used, ice water being used to saturate them at short intervals. After the disappearance of the symptoms of excitement, Priessnitz poultices may be applied. Internally sodium salicylate (15 to 20 gm. for large animals, and 1 to 2 gm. for small), or calomel (2 to 4 gm. or 0.05 to 0.4 gm. per diem except to ruminants) may be given.

If there are painful or tetanic contractions of the muscles, alleviation may be obtained by the administration of narcotics (chloral hydrate, potassium bromide, morphine).

In dogs lumbar puncture as described by Sabrazes and Muratet may be resorted to with the object of removing a portion of the cerebro-spinal fluid.

**Literature.** Fröhner, Monh., 1907, XVIII, 142.—Johne, S. B., 1896, 66.—Le Maitre, Rec., 1900, 529.—Marchand, Petit & Coquot, *ibid.*, 1906, 5.—Sabrazès & Muratet, Rev. Gén., 1906, VIII, 633.

## 2. Ossifying Inflammation of the Dura Mater of the Spinal Cord. Pachymeningitis spinalis ossificans.

There not rarely occurs in the dura mater of the spinal cord of dogs a peculiar inflammatory process resulting in the formation of plates of bone of various sizes which cause tearing and crushing of the roots of the spinal nerves, and even of the cord itself, and in addition may compress the cord.

**Historical.** Primary ossifying inflammation of the dura of the cord was observed by Mauri in 1878, Bonnet in 1881, Kitt and Stoss in 1883, and Fröhner in 1893. The specific nature of the disease was established by Dexler in 1893 and 1896 as a result of extensive clinical and microscopical examinations.

**Occurrence.** The disease is of comparatively frequent occurrence in dogs, but more rare than degeneration of the intervertebral discs. In three years, Dexler saw nine cases of this disease, and eleven cases of intervertebral enchondrosis. Single plates of bone, which caused no functional disturbance were found by Dexler in about ten per cent of adult dogs upon which postmortem examinations were made.

Siedamgrotzky found hard nodular elevations of a yellow color in the inner surface of the spinal dura mater of a tiger that was affected with paraplegia.

**Etiology.** No definite information can be given regarding the causes of the disease. Experience indicates that sex, age and breed have no influence on the occurrence of the disease, and at the most it appears to be more common in the larger breeds of dogs. As already indicated by Dexler, the actual cause should be found in the pronounced curvatures of the spine

that are sometimes present. In one case the disease was complicated with diffuse ossification of the vertebræ from the middle of the dorsal region to the sacrum, but there were no adhesions between the bones and the dura mater (Hutyra & Marek).

**Pathogenesis.** The hemorrhages and dilatation of the vessels found by Kitt and Stoss, and Cadéac, indicate that the disease is of an inflammatory nature. The plates of bone that are formed, press upon the motor and sensory nerve roots in their immediate neighborhood and, owing to the increase in thickness of the membranes, pressure may be exerted upon the spinal cord itself. They also hinder the free movements of the cord in the vertebral canal during changes of position of the body, and consequently there is a chance that the nerve roots, and even the spinal cord may be torn in the early stages of the disease. In time, the mechanical irritation in the cord and nerve roots sets up a chronic inflammation.

**Anatomical Changes.** Scattered along the dura mater from the cervical to the lumbar portions of the cord, and often in other parts, there are white or dark bluish-red plates of bone which by fusion may form quite large bony patches. In very severe cases, the dura may be converted into a hard tube. The majority of the plates are to be found on the ventral surface where they are almost exclusively situated in the early stages (fig. 96).

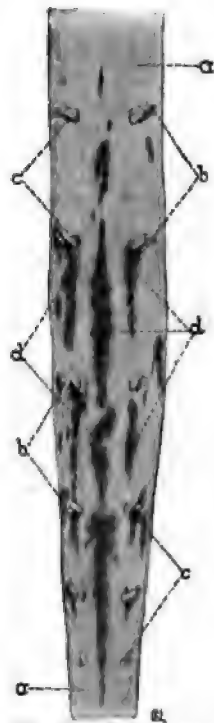


Fig. 96. Ossifying spinal meningitis. (a) Inner surface of the dura mater in the lumbar and sacral portions of the cord. (b) Sensory (c), motor nerve roots, at places surrounded by bony plates. (d) Plates of bone in the dura mater.

The plates develop on the inner surface of the membrane and are covered on the inner face with a thin layer of endothelium and on the outer with a thick connective tissue layer. They are composed of genuine bone tissue and show no tendency to unite with the vertebræ. There is an increase in the amount of connective tissue in the nerve roots and degeneration of the nerve fibers, while the lesions in the cord are less pronounced, but in severe cases are those of myelitis set up by pressure.

**Symptoms.** A variable length of time elapses before clinically recognizable symptoms make their appearance. According to Cadéac, this may be as long as one to two years after the onset of the disease. The first symptom observed is usually evidence of pain owing to the stretching of the sensory

fibres with every movement of the vertebral column. Animals sometimes cry out without any apparent cause, and still more when getting up, lying down, or making other movements. About the same time, but sometimes earlier, and at other times later, motor disturbances are observed. These may for a comparatively long time be due simply to pain or to muscular spasms caused by pain. The animal is easily fatigued, is unwilling to go up or down stairs and does so with difficulty, lies down cautiously and is very averse to moving the head or neck. During movement, the limbs are not flexed as much as normal, the gait is consequently more or less stiff, and in exceptional cases the animal may walk on its fore legs only. These symptoms may persist for a variable length of time or they may completely disappear, the animal appearing perfectly healthy during the interval.



Fig. 97. Maximal extension of the hind legs in ossifying spinal meningitis. (The specimen shown in fig. 96 was obtained from this animal.)

There may, however, be more striking symptoms. The muscular contractions become exaggerated, and owing to the increased tension of the extensors and adductors, the limbs are held in the position of maximal extension and adduction (fig. 97), and passive movements are possible to a limited extent or not at all. As a result of the compression of the motor nerve roots, and even of the cord itself, the symptoms of paralysis become more pronounced until in almost every case there is paraparesis or paraplegia, the paralysis being either symmetrically bilateral or appearing to be more severe on one side than on the other. The paralysis is most severe in the hind quarters, but in rare cases, it affects the fore quarters, but to a less extent. Paralysis limited to the fore limbs is possible



if the compression involves the brachial plexus only. The paralysis in most cases develops slowly, the first symptoms being a certain weakness of the hind quarters. This increases with periods of improvement until there is complete sacral paralysis, in which case the hind legs are dragged along. On the other hand, the case may get worse so rapidly that the animal appears to become paralyzed suddenly, but in such cases, the atrophy of the muscles indicates the slow development of the disease.

As the paralysis increases, the muscular tone decreases, and there is a rapidly progressive atrophy of the muscular tissue. If the paralysis is due to compression of the cord, the motor nerves remaining uninjured, there is no loss of muscular tone. In many cases there is a decrease in the susceptibility to electricity.

The hyperalgesia which, at the commencement, is evidenced by subjective symptoms, for a time increases as the motor disturbances progress. Light contact with the hair or a tap on the skin, etc., may cause symptoms of severe pain, while in other cases animals, which are obviously restless owing to paresthesia, bite their limbs until they are sore. Passive movements of the spine or percussion of the spinous processes in some cases cause symptoms of pain, but the position of the pain cannot be localized. In the later stages, the hyperalgesia may be replaced by hyperesthesia or complete anesthesia over limited or large areas.

At the onset of the disease all reflexes appear to be exaggerated. In many cases, muscular contractions may be caused by quite slight contact with the skin, scratching with the point of a needle, etc., and these may also involve the opposite side of the body. If the sole of the foot be scratched there is not simply a sudden flexion of the phalanges, but also an active extension of the whole of the opposite limb. Among the tendon reflexes the patellar reflex is most frequently increased, but in a large number of cases, active and very rapid contractions of the particular muscles are caused by percussion of the tendo Achillis, of the extensor tendons in the lower third of the fore arm or in the metacarpal region, the tendon of the tibialis anticus, the flexor metatarsi, and the distal tendons of the anconeus muscles. The priapismus which is sometimes observed affords further evidence of the increased reflex irritability, an erection being easily induced by rubbing the skin of the abdomen, the prepuce or the urethral bulb. If the finger be introduced into the rectum, the sphincter is thrown into repeated contractions and, as in stimulation of the abdominal skin, there is an involuntary and full flow of urine. This symptom must not be attributed to paralysis of the bladder.

In the later stages, reflexes disappear in those parts in which the motor and sensory nerves have lost their conductivity, while in cases in which there is compression of the cord



and the conductivity of the sensory and motor nerves is not destroyed there are active reflexes. In view of the fact that the process of ossification rarely extends backwards from the lumbar region to any marked degree, the reflexes in that portion of the hind quarters are often exaggerated in the later stages of the disease.

Incontinence of urine and involuntary discharge of feces is rarely seen in cases where the nerve roots of the bladder and rectum are involved, but somewhat more frequently if the caudal portion of the cord be compressed. Retention of both urine and feces is far more often seen. The digestive functions remain undisturbed for a long time, but finally nutrition suffers, and the animal becomes emaciated. In many cases, there are bedsores which render the animal liable to a general septic infection. Death is due either to an infection of this kind, to hypostatic congestion of the lungs, or to collapse.

**Diagnosis.** A suspicion as to the existence of the disease is raised if a dog occasionally, when changing its position or sometimes without obvious cause, utters cries. The following symptoms are characteristic of the disease: circumscribed but asymmetrical hyperalgesia of the skin, muscular spasms of the same and other parts of the body caused by passive movement of parts of the spine, symptoms of pain, the exact seat of which cannot be localized, in the later stages, loss of sensation in those parts which were previously hyperalgesic, paralysis of the muscles originally in a condition of spasms, ill defined areas that are in a condition of anesthesia, and, finally, symptoms indicative of pressure upon the cord.

The disease is most likely to be confounded with compression of the spinal cord, due to intervertebral enchondrosis, and as pointed out by Dexler a differential diagnosis is not always possible. The most important differential symptom is that in the last named disease the symptoms are localized up to the time of death, and indicate compression of the cord from the outset. In the ossification of the membranes, there is a gradual extension of the disease, and at first only individual nerves are involved, but later symptoms of chronic meningitis make their appearance. Compression of the cord is indicated when the area of sensory and motor paralysis is sharply circumscribed from the start, and the gait is consequently very unsteady; the paralysis in such cases is supranuclear. On the other hand, primary ossifying degeneration of the dura is indicated under the following circumstances: the reflexes which at the outset are very active, become gradually weaker, the muscular spasms which rapidly increase in severity are followed by relaxation and atrophy of the muscles, the area that is devoid of sensation is ill defined, and the disease is obviously progressive in character. Taking these and other points into consideration, it is quite possible to exclude spinal pressure.

Formerly, the disease was very frequently confounded with muscular rheumatism, but the distinction between the diseases is very striking in that in rheumatism there is no muscular atrophy, the reflexes are not affected, and the sensibility of the skin is normal. The disease may be distinguished from tetanus by the absence of trismus and spasms of the muscles supplied by the facial nerve. In spinal meningitis there is an absence of obvious objective pain, and owing to the more rapid development of the disease, muscular contractions are not observed. The course of transverse or diffuse myelitis is much more rapid.

**Prognosis.** When symptoms are already clinically recognizable, the condition becomes aggravated either gradually or suddenly until there is complete paralysis. In some cases, temporary improvements are seen.

**Treatment.** The anatomical alterations that are the cause of the disease exclude the possibility of radical treatment. Treatment can only be directed towards alleviating the pain, and where possible delay, the appearance of fatal symptoms.

**Literature.** Bonnet, Münch. Jhb., 1880-81, 109.—Dexler, *Ergebn. d. Path.*, 1896, III, 2, Abt., 516 (Lit.); *Die Kompressionsmyelitis des Hundes*, 1896.—Kitt & Stoss, *D. Z. f. Tm.*, 1883, IX, 142.

### 3. Injuries to, and Concussion of, the Spinal Cord. *Contusio et commotio medullae spinalis.*

**Etiology.** The spinal cord may be injured by the penetration of pointed objects entering the vertebral canal through intervertebral spaces, an accident which is especially likely to happen in the cervical region. An accident that occurs far more often is the crushing of the cord through dislocation of the vertebræ, owing to some violence, such as falling, being run over, collisions, etc. In many such cases, there is some fracture of the bones, the cord being injured by dislocation of the pieces or by splinters. In horses, and especially in well-bred animals, fracture of vertebræ occurs without any external violence, owing to severe muscular contraction, e. g., in jumping, kicking and galloping, or in attempts to get free after being cast. In many cases the resistance of the bones is decreased. Jacoulet and Vivieu proved the existence of osteoporosis in two cases of fractured vertebræ, and in many cases of fracture of other bones. Decreased resistance of the bone tissue must be assumed to be the immediate cause of fracture in cases in which there is osteomalacia, or loosening of the intervertebral joints owing to enchondrosis of the intervertebral cartilages. According to the observations of Fröhner, Platten, Nielsen, Pallain, and others, the injury causes only a splitting of the bone, the complete fracture developing after some time, generally some weeks.



Vertebral fracture occurs most frequently in the horse, and more rarely in other species. Tapken records several cases in cattle. The cervical vertebræ are most commonly involved, then the anterior lumbar bones, dorsal fracture being somewhat rarer. Powerful muscular contraction usually causes fracture of the lumbar or the most posterior dorsal vertebræ.

Dislocation and fracture of vertebræ cause hemorrhages between the membranes (intermeningeal hemorrhage), and in many cases, as observed by Schlesinger, hemorrhage into the tissue of the cord itself (hematomyelia). Similar hemorrhages may be caused by external violence bringing about no actual injury to the bone.

In rare cases, traumatism causes lesions which are only detectable by microscopic examination.

**Pathogenesis.** If the cord be crushed or subjected to pressure by extravasated blood, conductivity is destroyed both in the central and peripheral directions from the seat of the lesion. The conductivity may be destroyed completely, it may affect only one half of the cord, or a single column; nerve function will be abolished in the neighborhood of the lesion. The associated nerve roots are stimulated or destroyed by dislocation of the bones, and also by intermeningeal hemorrhage, which in some cases involves a large section of the cord.

**Symptoms.** In cases of complete cross section of the cervical portion of the cord anywhere between the medulla and the point of origin of the fifth and sixth pairs of cervical nerves, the animal dies in a few seconds, owing to the cessation of respiration, because the respiratory center is cut off from the nuclei of the nerve, supplying the muscles of respiration. During the brief period elapsing between the section of the cord and death, only the muscles of the head are moved. If the cord is not completely destroyed, the animal may remain alive for some hours, and exceptionally for several weeks, depending upon the extent of the injury. The symptoms presented are those of injury to the inferior segment of the cord and also in some cases bulbar symptoms (difficulty of swallowing, slowing of the pulse). The body temperature may be elevated (Schlesinger).

Total destruction posterior to the cervical thickening of the cord (posterior to the origin of the phrenic nerve) causes paralysis and loss of sensation in the extremities, body and tail. During inspiration, the thorax is not expanded, the ribs are motionless, and the respiratory movements are limited to the diaphragm, and through this the abdominal wall and the hypochondriac region (diaphragmatic respiration). In the fore quarters, the reflexes are destroyed but they persist and are even exaggerated in the posterior parts of the body. In-

voluntary passage of feces and urine, without incontinence, in other cases persistent retention of feces and urine, and sometimes priapismus may be observed. The pupils are equally or unequally dilated, but they react to light.

Injury to the dorsal portion of the cord leads to paralysis of the posterior half of the body. The reflexes are either normal or exaggerated, and the same disturbances of function of the bladder and rectum are observed as in the previous case. In exceptional cases, the contusion is limited to the anterior portion of the dorsal cord, in which case there may be diaphragmatic respiration.

Injury to the anterior portion of the lumbar segment of the cord causes paralysis and anesthesia of the croup, hind legs and tail. If the injury be in the middle portion of this region the symptoms are the same except that there is no patellar reflex owing to injury to the nucleus of the femoral nerve; the reflexes of the paralyzed portions of the body posterior to this are normal or increased. The same abnormalities with regard to the passage of urine and feces are observed as before. Destruction of the posterior third of the lumbar portion of the cord is generally associated with injury to the sacral cord and results in sensory and motor paralysis of the area supplied by the sciatic nerve, the nerves of the croup and tail, and the nerves supplying the sphincters of the anus and bladder. The anus remains open and stimulation of the sphincter does not close it. The urine trickles away.

At the moment of the injury, there are observed convulsive muscular spasms, which at first pass off very rapidly, but afterwards the contractions persist for a somewhat longer time. These are obviously due to a stimulation of the central motor path immediately before it is divided. Muscular contractions are observed either continuously or at intervals in the neighborhood of the injury owing to stimulation of the nerve roots, but in cases of extensive meningeal hemorrhage the contractions are for the most part observed in various parts of the body and the limbs.

In small animals, there is frequently demonstrable painfulness of the spine, in the larger animals, however, this is not as a rule observed. In cases of extensive meningeal hemorrhage, the areas showing increased sensibility vary in size because the sensory nerve roots are stimulated by the extravasated blood and the stimulus is carried towards the center by the uninjured cord. In a proportion of cases, there is a circumscribed swelling and crepitation, and very slight passive movement of the vertebræ can be noticed.

In the very rare cases of unilateral injury to the spinal cord, there is motor paralysis on the same side as the injury and sensory paralysis on the other.

In many cases the symptoms differ from those described. In some the paralysis and in others the anesthesia is less pronounced, or the reflexes posterior to the seat of injury are obliterated. Balint's investigations and the observations of Hutyrá & Marek on horses killed by pithing showed that the reflexes disappear behind the seat of injury if the roots or cells of the nerves in question were injured. In one horse that had fractures of the first and second lumbar vertebræ there was in addition to the symptoms due to the contusion great restlessness and difficulty of respiration, the cause being the simultaneous existence of an incarcerated diaphragmatic hernia.

**Course.** Even in those cases in which the injury does not cause the immediate death of the animal, death is the usual sequel. The larger animals die, as a rule, within one or two days, seldom more, but death is generally delayed somewhat in small animals, and results from bedsores, hypostatic congestion of the lungs or cystitis. Occasionally it happens that animals, and especially small ones, survive for a longer time, the symptoms persisting unaltered, or in cases where hemorrhage is the only lesion, gradually abate. Symptoms may disappear rapidly. The possibility of recovery does not appear to be excluded in cases in which the injury causes only hemorrhage or only slight lesions of the cord.

In some cases, the animals show no disturbance or only a slight paresis for some time, which may amount to weeks, after the occurrence of the injury, but then by some comparatively slight influence there is caused displacement of the ends of the bones and complete paralysis results.

**Diagnosis.** As in most cases there is an obvious connection between the symptoms of paralysis and some wound or injury, diagnosis usually resolves itself into determining whether the symptoms are due to some injury to the cord itself, or whether they are caused by hemorrhage. A point that is of value in coming to a decision is that disturbances due to hemorrhage only tend to improve with time, and also hemorrhage is indicated by a paralysis that is somewhat slow in development.

Dogs are often said to have been run over, when careful enquiry elicits the fact that the animal's movements were not quite perfect previously, and that there is either an ossifying inflammation of the dura, or there is some compression of the cord. In the differential diagnosis in horses the principal condition that is likely to be met with is paralytic hemoglobinemia. In this condition the paralyzed muscles are hard, the urine frequently contains blood pigment, and the sensibility of the skin is normal. In cases of pelvic fracture, there is no loss of sensibility of the skin, defecation and urination are not disturbed and there are symptoms indicating fracture of the bones.

**Treatment.** Improvement is only to be expected when there is simply contusion of the cord, owing to tearing of the membranes or intermeningeal hemorrhage. If in the larger



animals there is no improvement within a day or two, and if in the small animals there is an actual fracture or luxation of a vertebra, it is advisable to have the animal killed or sent to the butcher. If treatment be undertaken, the first essentials are perfect quiet and a soft bed. After some days, treatment may be commenced with systematic massage, followed by cautious movement, and, finally, electrical stimulation may be employed.

**Literature.** Arndt, *Ergebn. d. Path.*, 1903, IX, 1, Abt., 427.—Balint, *M. Orv. Arch.*, 1903, 1.—Dexler, *Ergebn. d. Path.*, 1900, VII, 466 (Lit.).—Knudsen, *Maanedsskr.*, 1909, XXI, 33.—Nielsen, *ibid.*, 1907, XIX, 1.—Pallain, *Vet. Journ.*, 1905, 259.—Petit & Desoubry, *Bull.*, 1905, 117.—Pilwat, *Z. f. Vk.*, 1902, 321.—Röder, *S. B.*, 1896, 137.—Tapken, *D. t. W.*, 1907, 489.

**Spinal Hemorrhage.** (*Apoplexia Spinalis, Hæmatomyelia.*) Hemorrhages of the spinal cord are generally due to inflammatory conditions or to injury, and consequently do not come in for further consideration here. There are very few references to spontaneous hemorrhage in the spinal cord in the domesticated animals; and they have no special clinical significance. Thomassen & Hamburger record a case in a horse in which there were small hemorrhages in the cervical and lumbar portions of the cord, and in which microscopical examination showed dilatation of the neighboring capillaries. This particular horse showed symptoms of paralysis which had disappeared the next day; but three weeks later there was paralysis of the bladder, tail and nerves of the face. The cause of the disease was in all probability inflammatory. Dernbach observed intermeningeal hemorrhage in a case of purpura. A case that was diagnosed as hematomyelia on purely clinical grounds by Lellmann was, judging from the manner in which it showed itself, and from its course, nothing but a case of pressure on the spinal cord.

#### 4. Inflammation of the Spinal Cord. Myelitis spinalis.

Under this term are usually included inflammatory conditions of the cord which may or may not be associated with softening, and which lead either to an infiltration with white corpuscles or to actual hemorrhage within the affected part of the cord.

**Occurrence.** The disease occurs most frequently in the dog, less commonly in the horse, and very rarely in the other species. In the dog the majority of cases occur in association with distemper, in the horse with influenza, apart from the so-called infectious spinal paralysis, which is probably inflammatory in origin (see page 697). Marchand, Petit & Bredo have recorded a case of cervical myelitis in a cock.

**Etiology.** Inflammation of the spinal cord is caused by viruses and bacterial toxins, and possibly also by other intoxications. The viruses of influenza, distemper, and rabies, often

cause inflammation of the spinal cord as has been frequently demonstrated in encephalitis. In very rare cases there develops simple tuberculous myelitis. It is remarkable that there is scarcely any tendency to purulent myelitis in the case of stranglers. There are also other unknown causes. In a case recorded by Dexler, a dog, eight years old, had got loose one cold night a month and a half previously and was found the next morning half numb. The dog showed symptoms of myelitis which were referred by Dexler to an infection of some sort. The same author observed a case of hemorrhagic inflammation involving the spinal cord as well as the brain in a horse. Marek records two similar cases in dogs, and one in a horse. Myelitis of unknown origin has been recorded in the horse by Weber and Barrier, Liénaux and Hendricks, Le Calvé, Watson, and Savary. Hutyra & Marek have on one occasion observed inflammation of the dorsal portion of the cord in a horse used for the preparation of diphtheria serum. Ceni and Besta produced chronic diffuse myelitis in a dog by feeding it on maize contaminated with the *aspergillus fumigatus*. The paralysis-like weakness observed in fowls by Wilke, and described by him as anterior acute poliomyelitis was probably a disease of the bones resembling rickets.

Marek found a diffuse inflammation of the spinal cord in four dogs extending over the greater part of the cord, and in all the cases there was extensive hemorrhage and necrosis of the cord, but he was unable to discover the cause of the condition. The possibility of its being distemper was excluded. In three of the cases extensive hemorrhagic enteritis was found at the postmortem, but it remained undecided whether the myelitis was a sequel to the enteritis or the enteritis a complication of the myelitis. Brown & Ophüls observed four cases similar to the above in dogs, but there was no enteritis.

As already mentioned, inflammatory processes involving the membranes frequently extend to the neighboring parts of the cord. The same thing happens in epizootic cerebro-spinal meningitis. On the other hand, diseases of the vertebræ and surrounding tissues rarely extend to the cord.

Some authors state that myelitis may be caused by cold or over-exertion, but these can only be considered as predisposing causes. Excessive use of animals for sexual purposes plays no direct part in the production of the disease.

**Anatomical Changes.** Macroscopic lesions are not always present. In cases of distemper-myelitis there are no hemorrhages or very slight ones, and usually also no softening, consequently the condition is often overlooked. In other cases, the inflamed parts of the cord are obviously altered. In recent cases they are softer, and in older cases, firmer. They are very rarely thickened, but more frequently become thinner. After section the substance of the cord generally runs out; the difference in color between the gray and white substance is indistinct, or there may be none. In acute cases the softened cord

substance appears moist and glistening, reddish in color and may be beset with hemorrhagic points. In certain cases (Brown and Ophüls, Hutyra & Marek) the cord substance is converted into a grayish-red pulp-like mass in which there is discoverable a reddish-brown track composed of extravasated blood. This can also be seen in neighboring parts that are apparently altered to a slight extent only. Pus is found very occasionally only in the cord, and when present it is generally the result of a direct injury. In acute cases there are often sharply circumscribed areas of hyperemia in the membranes, and even inflammatory alterations. In chronic cases there may be thickening of the membranes.

Errors of diagnosis are possible if a thorough examination of the cord is undertaken, and principally because, however carefully it may be taken out, it is scarcely possible to avoid cutting it and the cuts which pass unobserved cause a softening of the substance of the cord and this may be thought to be due to inflammation.

Information as to the nature and extent of the lesions can only be obtained by histological examinations. In acute cases the blood vessels appear to be dilated and there is a cellular and in some cases a fibrinous exudate round about them. In many cases the hemorrhagic character of the inflammation is pronounced, the perivascular spaces being occupied by large groups of red corpuscles, or, as in the cases of diffuse hemorrhagic myelitis observed by Brown & Ophüls and by Marek in the dog, masses of red cells are visible occupying large sections of the cord, the surrounding nerve tissue appearing necrotic. In the nerve cells are to be found all stages of chromatolysis, peripheral disposition of the nucleus, shrinking and disappearance of the cell processes, and even of the cells themselves. There may also be varicose swelling of the axis-cylinder processes, degeneration and destruction of the medullary sheaths. The diseased parts are rich in cells containing fat. At places where the nervous tissue is destroyed there may be a proliferation of the neuroglia, but in chronic cases there may be a production of fibrous connective tissue and a thickening of the vessel walls.

In cases in which the inflammation involves the whole thickness of the cord the centripetal paths (posterior columns, lateral cerebellar tracts, Gower's tract, individual fibers of the lateral tract) undergo degeneration towards the brain, and in the centrifugal paths (central motor paths, and, as shown by Dexler and Liénaux, Schultze's tract in the basal portion of the posterior columns) the degeneration proceeds in the opposite direction. In cases in which the inferior cornua are diseased the inferior nerve roots and the motor fibers of the nerves involved also degenerate.

The disposition and extent of the lesions varies from case to case. The spinal cord may be involved through its entire thickness, or for a short distance only (myelitis transversalis), or it may be very extensively diseased, and in still other cases there may be large inflamed areas which do not involve the whole thickness of the cord. In such cases, the lesions are scattered through the tissue (myelitis disseminata). It is very exceptional to find a single inflammatory center (myelitis focalis).

**Symptoms.** In cases of *myelitis transversalis* the nerve cells at the seat of the inflammation are killed and the tracts which pass through it on both sides of the cord are interrupted. Consequently, there is a peripheral paralysis in the part of the body involved and a central paralysis in parts lying posterior to it, associated with loss of sensation (paraplegia, para-anesthesia). It is easily understood that the



peripheral paralysis is only recognizable in cases in which the inflammation is somewhat extensive; not rarely only the central paralysis is observed. The anterior limit of the area that is devoid of sensation coincides with the anterior limit of the inflammation, but it appears to be displaced a little posteriorly owing to the distribution of the sensory nerves, the displacement being greater the more posteriorly placed the inflammatory lesion is. The numbed area is generally separated from the normal tissues by a zone of varying width that is in a condition of hyperesthesia.

The sensory and motor disturbances reach their maximum after some hours, days, or even weeks. At first, the only symptom is that the animals tire rapidly, they lie down frequently



Fig. 98. Spinal meningitis with complete paralysis of the hind quarters.

and remain lying for long periods, and rise with difficulty. Very soon the gait becomes trailing, the joints of the limbs give way and are placed sometimes in positions of abduction and sometimes in positions of adduction. The body is not brought forward sufficiently and sways about. After a time there is complete paralysis. When this has occurred, the animal can no longer get up or move his limbs. Small animals drag the hind quarters along on the extended hind legs (fig. 98), provided the seat of the inflammation be posterior to the cervical thickening of the cord.

When the myelitis transversalis develops rapidly, the muscles of the paralyzed parts of the body are relaxed, but if the course of the inflammation be slower, there is a certain amount of muscular rigidity in those parts of the body that are posterior to the seat of disease.

The tendon and skin reflexes in those parts of the body that are involved in the supra-nuclear paralysis, are as a rule, exaggerated. But if the myelitis be more extensive, the reflexes at the anterior limit of the paralyzed area are either reduced or completely absent. The muscles in this area respond to a sudden blow with a slow and sluggish contraction

(mechanical reaction of degeneration), which is frequently absent if the blow be repeated a number of times, but after a pause reappears.

Provided the intra-muscular nerves remain intact they respond to mechanical stimulation of the muscle belly with a contraction caused by their excitability. If there is already degeneration of the nerve, especially after destruction of the cells of the inferior horns or of the peripheral nerves, their excitability is lost and under these circumstances the muscle fibers which are extremely excitable will give only a slow contraction which is due exclusively to the mechanical stimulation and is produced entirely without nervous influence.

In cases where the spinal cord exclusively is involved, there is no pain. In exceptional cases, the insensitive area is margined by a hyperesthetic zone, this is due to secondary spinal meningitis.

As a rule, there is no ataxia. One can only conclude from the severe disturbances of locomotion, which are not proportional to the paresis, and which increase with the loss of the power of vision, that there is simultaneous ataxia, provided that there is no great loss of muscular power.

If the disease involves the sacral portion of the cord, the urine trickles away, and there is involuntary defecation, but if the disease affects higher portions of the cord, retention of urine and feces is observed, copious discharges of urine taking place at times.

Among the trophic disturbances the simple muscular atrophy which is caused by persistent supranuclear paralysis, must be mentioned. Towards the end of the first week there may be muscular atrophy associated with degeneration reaction in the area involved in the nuclear paralysis, provided the inflammatory changes are extensive (Hutyra & Marek). In some cases there is edema, and now and then, localized sweating is observed.

The symptoms of inflammation of the various portions of the spinal cord agree in the main with the symptoms produced by contusion of the same portions (see page 686). If the inflammation extends to the medulla and if the connection between the brain and the respiratory center is not completely interrupted, serious symptoms may make their appearance, such as difficulty in swallowing, irregularity of the heart, which is sometimes accelerated, and sometimes slowed, irregular respiration, etc., and finally, death may occur suddenly owing to paralysis of the respiratory center.

**Diffuse myelitis** generally involves a circumscribed portion of the cord and extends rapidly either upwards or downwards (Myelitis ascendens, M. descendens). In occasional cases, the area originally involved is large. In the majority of cases it commences in the more posterior parts of the cord, and within a short time paralysis and loss of sensation of the tail, the croup, and the hind legs occur, associated with incontinence of urine, involuntary defecation and relaxation of the sphincter of the



anus. The anterior limit of the affected area soon moves in the forward direction and reaches the abdominal wall, and from there extends to the thoracic wall, respiration being carried on solely by active contraction of the diaphragm. Shortly after this there is paralysis of the muscles of the fore limbs, and the animals lie on the ground helpless but perfectly conscious. Death occurs shortly after, owing to the diaphragm becoming involved. The groups of muscles which lie immediately in front of the progressing part of paralysis show, as a rule, fibrillar twitchings and even slight clonic and tonic spasms before they become paralyzed. The animal suffers from hiccoughs before paralysis of the diaphragm occurs. The paralyzed muscles are in a condition of complete relaxation, and offer no passive resistance. The reflexes are completely destroyed in the paralyzed area, but percussion of the paralyzed muscles produces slow contractions right up to the time of death (mechanical degeneration reaction).

If the process starts higher up and spreads further forwards or backwards, symptoms of supranuclear paralysis are observed from the outset in the latter case in the parts of the body posterior to the seat of the inflammation, this gradually passes into a nuclear paralysis with obliteration of the reflexes and paralysis of the sphincters. If the process extends forwards, the original paralysis remains supra-nuclear, but towards the head the nuclear paralysis involves an ever increasing area.

In **myelitis disseminata** inflammatory foci occur in both the white and gray matter of the cord, the distribution and extent of which are variable, and in consequence the symptoms vary from case to case. The disease is usually seen in cases of distemper, and it is apparently not rare to observe hemorrhagic myelitis in the horse. In this as possibly also in other forms of myelitis there may be centers of inflammation in the brain, in which case the symptoms would be still more complicated.

In the majority of cases of myelitis disseminata due to distemper, there are more or less rhythmic contractions which generally involve the muscles of the extremities, but more rarely may affect the muscles of the neck or the abdominal muscles exclusively. The muscles of the chest and the muscles supplied by the radial and sciatic nerves chiefly are involved. In the majority of cases there is a nuclear paralysis, and sometimes also a supranuclear paraplegia of certain groups of muscles of the extremities and of the sphincters. Charitat observed persistent gnawing of the end of the tail (automutilatio) in a dog that was fully conscious.

In a case of disseminated myelitis in a horse Weber and Barrier observed a gradually progressive paresis of the extremities and a consequent rapid tiring of the patient. Three months later the disturbance was more striking. During motion both the fore and hind feet were lifted jerkily, abducted and advanced with excessive flexion, adducted and set down clumsily. This was followed by marked



flexion of all the joints under the body weight, the animal stumbled frequently and staggered to such an extent that it was likely to fall at any moment. Blindfolding the eyes exaggerated the motor disturbances to a marked extent, showing that ataxia was also present. That the motor disturbances were not due exclusively to ataxia was shown by the fact that there was also paresis, the horse being unable to hold back a cart when going downhill. The muscles on the anterior surface of the upper arm and the superficial croup muscles were rigid, but the extensors of the elbow and the posterior muscles of the croup were relaxed. After having been under observation for nine months the animal was killed. In the right half of the spinal cord, immediately in front of the seventh nerve there was an inflamed area about  $1\frac{1}{2}$  cm. in length involving the outer third of the posterior column, the lateral column, and the greater part of the gray matter. In the left half of the cord in the lumbar region there were three similar centers situated close together and involving the greater part of the lateral column and the superior horn.

In a case recorded by Dexler the disease commenced with paresis of the off hind leg and of the corresponding fore leg, partial facial paralysis, and sharply defined hyperidrosis of the same side. Two days later there was paresis of the other legs. The muscles of the off fore leg were rigid and those of the hind relaxed. Numerous centers of inflammation were found scattered irregularly in the white and gray matter in the cervical and dorsal portions of the cord.

In a case in a horse recorded by Hendricks and Liénaux there was staggering gait, jerky lifting of the foot as it was brought forwards, dragging of the wall of the off fore foot along the ground, loss of sensation of certain parts of the fore and hind legs. Blindfolding did not aggravate the disturbances. In the cervical region, about opposite the seventh vertebra, there was a sclerotic area with ascending degeneration of the posterior column. In the dorsal portion of the cord about opposite the first vertebra some small irregular foci were also found.

In a case of cervical poliomyelitis in a cock Marchand, Petit and Bredo observed paresis and hyperesthesia of the wing, and lateral flexion of the neck during repose so that the beak was directed backwards. During feeding there was no abnormality of the position of the head.

**Course.** The duration of the disease is influenced principally by the seat of the inflammation and the rapidity with which it spreads. Both transverse and diffuse myelitis run a short course especially in the larger animals, death taking place within a few days or in very rare cases a little longer. In the case of disseminated myelitis on the other hand, if there is no extensive paralysis the disease may last for a month and even longer. In such cases the disturbances become more severe slowly and spasmodically, and sometimes come to a halt, a condition of chronic myelitis having been produced in the meantime. Provided no febrile complications occur, there is no rise of temperature, even in the acute cases. Death is caused directly by general sepsis, cystitis or enteritis, and in many cases by cessation of respiration.

Recovery in the clinical sense of the term is exceptional, as for example in cases of distemper, in which the inflammation is not very extensive. Not rarely some improvement is observed, but generally a sudden exacerbation occurs after some time.

**Diagnosis.** A diagnosis may be based upon the presence of the symptoms above described, which have no obvious cause or no association with the diseases mentioned in the paragraph relating to the etiology, upon the rapid progress of the condition, and the absence of objective symptoms of pain in the vertebral column and the skin.—The differential diagnosis of acute

myelitis and ossifying myelitis has already been dealt with (see pages 679 and 684).—In cases of traumatism of the spinal cord, there is in most cases some evidence of the mechanical interference. Symptoms appear suddenly, and there is often evidence of pain in a circumscribed portion of the spine, distortion and crepitation.—Compression of the cord is usually characterized by a slowly progressive paralysis associated with active contraction of the muscles; sensation persists for a longer time, and is not destroyed in the majority of cases even in the later stages. There may be pain and distortion of the spine in the late stages. There is great difficulty in excluding cases of compression of the cord in which the symptoms of paralysis are actually or apparently produced suddenly, or in which there is neither pain nor distortion. Recognizable circumscribed atrophy of the muscles, the repeated appearance of these or similar symptoms, stiffness of the paralyzed muscles, and a disappearance of the paralysis in the later stages contraindicate inflammation of the spinal cord. In the dog, compression of the cord is the most commonly observed disease of that structure.—Diseases of a general nature associated with pronounced muscular weakness are still often confounded with myelitis. There is a special tendency for the hind quarters to appear weak in certain diseases of the intestine or peritoneum that are associated with pain in the smaller animals and these symptoms are often said to be due to disease of the spinal cord without further investigation. In these cases, disease of the cord can generally be excluded if the sensibility of the skin and the reflexes are investigated, defecation and micturition are carefully observed, and a thorough examination of the other organs is made.

**Treatment.** Bedsores are avoided by the provision of a soft bed and the frequent turning of the animal. Frequent grooming prevents the development of dermatitis. If there is retention of the urine and feces, the bladder may be evacuated by pressure with the flat of the hand, or by the use of the catheter, and the feces may be removed manually.

Internally, potassium iodide in doses of ten to twenty and 1 to 2 gm. is administered in most cases, but no special results follow its use. If muscular atrophy should threaten or be actually present, attempts should be made to check it by passive movements of the paralyzed limbs and massage. This treatment promises best in cases of supranuclear paralysis. It is absolutely useless to administer nerve stimulants such as strychnine (0.05 to 0.10 gm. or 0.001 to 0.003 gm.), veratrin (0.05 to 0.20 gm. or 0.01 to 0.02 gm.) subcutaneously, arsenic (liquor arsenicalis 10 to 15 gm. or 2 to 5 drops daily per os). Many authors have recorded rapid improvements following the subcutaneous injection of eserine, pilocarpine or arecoline in dogs. It is possible that in such cases the pressure on the spinal cord

is due to degeneration of the intervertebral discs, a condition which frequently improves rapidly without any treatment whatever. In the small animals, electricity may be employed, a large electrode being placed on each side of the spine, and a strong galvanic current passed for thirty minutes. The paralyzed muscles may be stimulated by faradization, or if their sensibility to this be lost, a galvanic current may be used. But no more is to be expected from electricity than from other methods of stimulating the muscular, nervous and circulatory systems.

In cases in which any of the above treatments are without avail, animals that are fit for food should be sent to the butcher as soon as practicable.

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## 5. Enzootic Spinal Paralysis of the Horse. Paraplegia enzootica equorum.

This is, as a rule, an acute general infectious disease characterized by multiple capillary hemorrhages in the various organs and especially in the spinal cord. There is frequently a gelatinous infiltration around the bladder and the genital organs. It is caused by a specific streptococcus and its special clinical feature is paralysis of the loins.

**Historical.** The disease was first observed and accurately described by Comény in France in 1888. Further additions were made to the literature upon the subject by Mulatte (1897), Grange and Mégnin (1897), Blin and Lambert (1897), and Sorriau (1905). In Germany, it appears to have been recognized as a specific disease by Schmidt (1885), and by Albrecht (1896). A contagious lumbar paralysis of the horse in India (Kumri) was described by Burke (1897). The etiology of the disease was investigated by Schlegel (1906), Zwick (1907), and recently by Perucci (1910). The disease was connected with paralytic hemoglobinemia by Schlegel, as indeed it had already been by earlier authors.

**Occurrence.** The disease appears to have a very localized distribution and occurs only in certain stables. In these stables, there may be an alternation of contagious cases with cases that appear to be more sporadic in nature.

Repeated outbreaks have been observed in Germany by Schlegel, for instance in Prussia, Saxony, Baden, Württemberg, and Hesse.

In France it has been observed as an epizootic among army horses.



According to Dahlström a contagious lumbar paralysis has been known in Denmark and Sweden for more than 50 years, which is probably the same disease.

Christianssen and Rasmussen observed weakness of the hind quarters in foals from 1 to 2 years old which lasted for months and even 1 to 2 years.

In Austria a similar disease has been observed among the army horses in the neighborhood of Vienna. This was considered by Szerdahelyi to be due to alkaloid poisoning.

Small outbreaks have been recorded in Italy by Perucci in Bologna.

**Etiology.** A streptococcus which is noncapsulated has been described as the cause of the disease by Schlegel, Zwick and Perucci; and Schlegel has named it streptococcus melano-genes. The organism can be found in the blood, parenchymatous organs, bone marrow, spinal cord, the gelatinous infiltration around the bladder and genital organs and in the urine. In the circulating blood it occurs either as mono- or diplococci, or in short chains, but as a general rule the chains are longer. These long chains are prominent in the acute cases.

**Staining.** The streptococcus stains easily and well with the basic anilin dyes, and according to Zwick and Perucci, is Gram-positive; but according to Schlegel, it is Gram-negative.

**Cultivation.** The organism is an aerobe and the optimum temperature for growth is that of the body. The best growths are obtained on media containing defibrinated rabbit or horse blood. In broth colonies develop in large numbers in 24 hours. These settle to the bottom or on to the sides of the vessel, the liquid remaining clear. Perucci observed a rapid decrease in the profuseness of growth in simple broth cultures and also on other media containing no blood. In broth containing blood, growth is abundant, and the colonies which fall to the bottom appear brownish-gray in color, owing to decomposition of the blood-pigment. In agar growth takes place along the needle track in the form of a fine grayish-white thread with down-like outgrowths. On blood-agar the organism produces pale grayish-white colonies surrounded by a deep brown zone, resulting from the decomposition of the blood by the streptococci. Nieter found the last-mentioned characteristic, common to many other streptococci.

**Pathogenicity.** The organism isolated by Schlegel causes a general infection in the small experimental animals, characterized by paralysis of the hind quarters, and other lesions similar to those seen in a natural case in the horse. Perucci, however, was able to infect rabbits only by intravenous, intraperitoneal, and subcutaneous inoculation with blood or culture, the inoculation setting up a general disease, associated with degeneration of the parenchymatous organs. In the horse, Schlegel observed only a temporary weakness of the hind quarters after the intravenous introduction of large quantities of culture. Zwick produced the typical disease by repeated injections, and Perucci succeeded with a single intravenous inoculation of 10 cc. of broth culture. The administration of cultures by the mouth caused only a transitory paresis in a horse (Zwick), while intraperitoneal inoculation caused a slowly progressive lumbar weakness without hemoglobinuria, a sero-purulent peritonitis, and lesions similar to those seen in a natural case (Schlegel).

So far the **natural infection** is not known. Schlegel supposes that the streptococci maintain a saprophytic existence in the intestine of otherwise healthy horses, and under certain

favorable circumstances, such as feeding on maize, bran, beets or potatoes, after overexertion, faulty attention, the presence of parasites, assume pathogenic properties. The diseased horses can infect others, because the streptococci which have become pathogenic are passed out with the feces and urine, and should these come into contact with food or water or be introduced into healthy horses in any other way, infection results. By feeding a horse, a donkey and a male goat on some suspected hay Zwick succeeded in causing the typical disease, although under natural circumstances the disease appears to be confined to the horse. Comény and Sorriau are inclined to think that infection takes place by way of the urethra and the urinary tract. On the other hand, Kull and Duvinage observed the disease in connection with an infectious catarrh of the upper air passages.

**Pathogenesis.** From the experiments that have been made up to the present the only conclusions that can be drawn are that the streptococcus causes a general infection, associated with hemorrhages and parenchymatous degeneration of various organs. No proof has been furnished as to the inflammatory nature of the lesions in the bone marrow and the central nervous system. Similar extravasations occur in any case in which there is dissolution of the blood. An explanation is necessary as to what extent the symptoms of paralysis are due to the hemorrhages in the spinal cord on the one hand, or to the lesions in the long bones and the resulting pain on the other, or whether they may be caused by the septicemic infection.

**Anatomical Changes.** The peritoneum shows petechial hemorrhages. The mesenteric glands are markedly swollen and infiltrated with blood-stained serum. The spleen is normal or it may be enlarged three or four times. The liver and kidneys are enlarged and show parenchymatous degeneration and hemorrhages. The bladder often contains urine stained with blood, the mucous membrane is beset with hemorrhages and the wall somewhat thickened. In a number of cases the connective tissue around the bladder and genital organs is gelatinous and there may be edematous swelling of the lips of the vulva or of the sheath, and sometimes of the skin of the perineum. The mucous membrane of the vagina is in such cases studded with small hemorrhages. The marrow of the long and flat bones is stained brown or brownish-red, either diffusely or in patches, owing to hemorrhages, the appearance resembling that seen in infectious anemia (see Vol. I). There is a blood-stained fluid in the epidural and subdural spaces, the arachnoid and the pia mater are swollen and gelatinous, and the latter appears to be beset with small hemorrhages. In the spinal cord there are numerous minute hemorrhages and reddish-yellow softened centers, especially around the central canal, which will

probably be only recognized by microscopic examination. There is no cellular infiltration (Perucci). In some cases there are no macroscopic lesions.

**Symptoms.** Clinically the disease, according to Schlegel, passes through an occult and an open stage, while Perucci distinguishes between a peracute, an acute and a subacute form. The subacute form described by Perucci corresponds with the occult stage of Schlegel.

In the occult stage there is emaciation, weakness, languor and loss of power in the hind quarters, the gait being uncertain in consequence. Stumbling, paddling with the hind feet, cramp-like contractions of the muscles of the loins, croup and abdomen are observed frequently. In some outbreaks there is slight edema of the sheath and vulva, in which case there is often a sediment in the urine.

The open stage commences with collapse of the patient during work or movement, or in some cases while the animal is at rest (peracute form). In many cases the lumbar paralysis develops in stages and is complete within some hours or two or three days (acute form). The animal is only able to get up with help or not at all. In cases in which the paralysis is not yet complete the animals paddle with the hind feet, keep getting up and lying down again, and groan. The appetite is not disturbed and, according to Perucci, there is no elevation of temperature. By this time bile-staining of the conjunctiva is usually recognizable and shortly after a rise of temperature ( $41^{\circ}$  to  $42^{\circ}$  C.) and a notable acceleration of the pulse. There is strangury and the urine often appears red in color and contains albumen, red blood corpuscles and bile pigments. There are no obvious sensory disturbances and there is no paralysis of the sphincters; the only authors to record these symptoms being Duvinage and Perucci. In the later stages of the disease there is marked emaciation.

In certain outbreaks, inflammatory lesions of the external genital organs have been observed. The penis hangs out of the sheath which is swollen and edematous, its outer surface being beset with minute hemorrhages; the lips of the vulva gape and the mucous membrane of the vagina is intensely red in color. Sometimes the edema extends to the perineal region.

**Course.** This differs in different outbreaks. The duration of the disease may vary from one or more days to a week in some cases, and in others from several weeks to three months, so that an acute, a subacute and a chronic course may be distinguished. According to Schlegel the disease is fatal in the majority of cases (70 to 100 per cent), but other authors have recorded recovery in 50 per cent of cases, especially in the occult stage of the disease. Convalescence is slow.



**Diagnosis.** A diagnosis may be based upon the symptoms of paralysis unassociated with any special sensory disturbance, the maintenance of the appetite for some time, the absence of hemoglobinuria, the rigidity of the muscles of the quarters, in many cases the edema of the genital organs, and on the infectious nature of the disease in many outbreaks. In sporadic cases, and especially during the occult stage of the disease, confusion is possible with other diseases. Whether, as believed by Perucci, a diagnosis may be based with certainty in such cases upon the cultural demonstration of the causal organism in the blood is a matter for further investigation. The conditions that are likely to be confounded with the disease are infectious anemia, paralytic hemoglobinemia, piroplasmosis (see Vol. I), and, in the postmortem room, sclerostomiasis (see page 488). Septicemic conditions and certain intoxications must also be excluded.

**Treatment.** This is useless in cases that develop rapidly. In less acute cases the treatment advised for paralytic hemoglobinemia (see Vol. I) may be applied. Symptomatic treatment and the administration of disinfectants must also be considered. In a case of the authors, atoxyl produced no satisfactory results (see page 492).

As prophylactic measures Schlegel advises that a diet containing large proportions of maize, bran, molasses, roots or potatoes should be avoided, and that the stables should be kept clean and well ventilated. Healthy horses should be separated from the diseased, and intestinal disinfectants should be administered. A change of diet appears to be indicated.

According to Perucci the injection of anti-streptococcic serum is useful in outbreaks inasmuch as the animals acquire an increased resistance.

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**Tabes Dorsalis.** Although this disease occurs frequently in man in connection with diseases of the spinal cord, it so happens that tabes dorsalis or an analogous disease does not occur in animals. Tabes dorsalis (progressive locomotor ataxia of Duchenne) is a quite definite chronic disease of the central nervous system, the most striking feature of which is a degeneration of the posterior columns of the cord, involving the posterior nerve roots, and also to a greater or less degree the other centripetal paths. There is also degeneration of certain groups of fibers in the brain. The disease is systemic and tends to involve constantly certain groups of fibers.

In veterinary practice, cases have been and are still reported which

are said to prove the occurrence of *tabes dorsalis* in the lower animals. For a long time trotting disease (*Traberkrankheit*) of the sheep, and the nervous stage of dourine were thought to correspond with *tabes* in man, until histological examinations finally showed that there is no connection between them. Fröhner claims to have seen cases in the dog exactly simulating *tabes*, Liénaux and Hendricks record a case in a horse that closely resembled *tabes* both clinically and histologically; and Hendricks records a case of locomotor ataxia in a dog. Hamburger found degeneration of the posterior columns in a dog, but the case was not observed clinically.

These and similar cases do not prove in the least the existence of *tabes* or a disease like it in animals. The cases that were observed, only clinically furnish no proof because the diagnosis was based simply on the symptoms of sensory disturbance, and sufficient consideration was not given to the fact that similar disturbances may be caused by other lesions of the motor paths, as components of the co-ordinating system (see page 588). Besides the condition of the reflexes is not mentioned, and in cases where such examination was made (Hendricks) they were found to be exaggerated. No histological evidence of the occurrence of the disease in animals has been adduced. The degeneration of the posterior columns observed in sheep by Anacker in cases of trotting disease have not been found by investigators working with accurate methods. The case of Liénaux and Hendricks, already mentioned, merely shows that in this particular case there was disseminated myelitis, which happened to produce a lesion in the cervical portion of the posterior columns with consequent degeneration upwards. It is merely a case of localized disease of the cord associated with the not unusual ascending degeneration. Hamburger's case in the dog was similar in nature. Hutyra & Marek have found pronounced degeneration of the posterior columns in cases of disseminated myelitis in distemper; but there was not the slightest resemblance to *tabes*.

## 6. Compression of the Spinal Cord. *Compressio medullae spinalis*.

Compression of the cord is caused by a variety of diseases in which there is a reduction in the lumen of the vertebral canal.

**Etiology.** The primary disease may involve either the tissues immediately surrounding the vertebral column, the vertebral column itself, the vertebral canal, the membranes of the cord, or the cord. The following diseased conditions may cause compression of the cord.

**Degeneration of the Intervertebral Discs** (*Enchondrosis intervertebralis*). This occurs almost exclusively in the dog and was first described accurately, clinically and histologically by Dexler. Recently two cases have been observed in the pig by Marek. Nothing definite can be said as to the cause. The frequency of the occurrence of the disease increases with age. The occurrence of the degeneration in those parts of the spine that are the most mobile appears to suggest that it may in some cases be caused by excessive curving, strain, etc., of the spine,



and especially in breeds of dogs in which the spinal column is too long in comparison with the length of the animal's legs. This was shown by Jakob regarding the striking disproportionate length of the body and the limbs in the case of dachshunds. The prejudicial effect of this body-formation is increased by the weakness of the joints of the limbs. A few observations have shown that the joints that are affected most commonly are the tenth and thirteenth dorsal, the first and fourth lumbar, and somewhat more rarely the second and fourth cervical. It is very rarely that other joints are affected. According to Dexler the cervical vertebræ are involved as frequently as the dorsal and lumbar joints, and Cadéac believes that the disease occurs principally in the cervical vertebræ. It is rare that a single joint is affected. In cases in which the posterior dorsal vertebræ are involved the lumbar bones are also affected, and, similarly, disease in the cervical vertebræ is associated with disease in the dorsal bones. The disease is not, as a rule, co-extensive in all the joints.

Dexler's histological investigations have shown that the disease begins with the formation of a cellular tissue richly supplied with vessels in the periosteum of the vertebra and the intervertebral discs, the tissue in the latter case containing islands of hyaline cartilage. Later the number of vessels decreases, the connective tissue shrinks, and the islands of cartilage increase in size and finally become calcified.

At the postmortem there are found, both in the vertebral canal and also on the outer surfaces of the vertebræ, white prominences which at first are soft, but afterwards become bony. In the later stages the bony growths within the canal, which are boatlike in shape, extend from one vertebra to the next and form a connection between the two. As a result of persistent irritation, in cases that have been in existence for some time a chronic inflammation of the dura is set up in the immediate neighborhood. This generally leads to the production of new cartilaginous tissue which calcifies subsequently. In time the thickened portions of the membrane become joined to the intervertebral growths and so further reduce the lumen of the canal (fig. 99).

Whether the process described in the intervertebral discs is actually inflammatory or of some other nature is not definitely known, but the inflammatory nature



Fig. 99. Projection of the intervertebral discs into the spinal canal owing to enchondrosis intervertebralis. (a) Between the second and third cervical, and (b) between the eleventh and twelfth dorsal vertebræ. At (a) the dura was adherent to the prominence.



of the change is indicated by the histology of the lesion. That the condition is inflammatory is also indicated by the facts that in some cases there is a simultaneous ulcerative inflammation of some of the joints of the limbs (Hutyra & Marek), and that Cadéac has observed exostosis-formation in the joints of the limbs in many cases.

**Intervertebral Ossification** (*Enostosis intervertebralis*). In old horses and dogs there sometimes develop, as a result of an osteoplastic periostitis in the neighborhood of the intervertebral discs, growths composed of true bone. These growths vary in size and project into the vertebral canal and join the opposed parts of two or more joints by means of closely set toothlike projections, so firmly that the whole spine with the exception of the neck is converted into a solid column. The process occurs in nine-tenths of the cases in the most mobile parts of the dorso-lumbar sections of the spine, that is, the parts which are espe-



Fig. 100. Vertebral tuberculosis in a pig. (a) Tuberculous growth in a dorsal vertebra. (b) Spinal cord showing "hour glass" compression.

cially exposed to strains. Similar growths may also result from fracture of vertebræ (Cadéac, Dexler, Ryder, Hutyra & Marek.

**Ossifying Pachymeningitis** (see page 680). This condition in some cases leads to compression of the cord.

**Tuberculosis.** Both in cattle and in the pig tuberculosis is frequently responsible for compression of the spinal cord. A similar case has been recorded by Mégnin in the horse. Tuberculosis of the bones occurs most frequently in the dorsal portion of the spine, and more rarely in the cervical and lumbar regions. Of eleven cases collected by Hamoir, seven involved the back, two the neck and two the lumbar bones. The disease leads to the production of softened or caseous centers, or in some cases to the formation of sarcomalike masses with considerable enlargement of the vertebral bodies, or more rarely the

vertebral arches (see fig. 100). Tuberculous meningitis is usually seen in the dorso-lumbar portion of the cord, and more rarely in other parts, or even throughout the length of the cord. This leads to the production of a varying number of tuberculous nodules of different sizes with caseous centers. Round about these nodules there are often lesions of a more acute nature. Tuberculosis of the spinal membranes is of very rare occurrence, and so far has been observed in the form of caseous or calcified centers in the lumbar portion of the cord only (George and Johnne, Kitt, Steuding, Schmidt, Hamoir). One case has been observed in the dog by Poulin and one in the pig by Vogt.

In a few cases **actinomycosis** of the membranes has been observed in cattle (Mathiensen, Koorevaar, Poes). In such cases there was enlargement of the vertebræ with the formation of cavities full of pus. According to Mathiensen the lesions are found in the anterior parts of the neck and back.

**Neoplasms** are rarely the cause of compression of the cord. In many cases they develop in the neighborhood of the vertebræ and, penetrating through the intervertebral foramina, or through the vertebræ themselves, gain access to the canal (Dörrwächter, King, Hertwig, Petit, Hutyra & Marek). In other cases they develop in connection with the bones, the membranes, and, in very exceptional cases, the cord itself. In cases of general melanosis in the horse, and especially in gray horses, melanomata develop in connection with the vertebræ, and in very occasional cases, exclusively in the meninges. These growths appear to be found most commonly in the dorso-lumbar region. Sarcomata have been met with in the horse, ox and dog, gliomata and glio-sarcomata in cattle, (Kitt and Dörrwächter), papillomata and sarcomata occasionally in the horse, lipomata principally in cattle, cholesteatomata in the horse and dog (Dexler), and finally, chondromata in horses. From a clinical point of view, inflammatory tissue produced by injuries (hemorrhages, fissures in bone; Fröhner, Rubay and Navez) is as important. Exceptionally aneurism of the aorta causes erosion of the bodies of the vertebræ, and compression of the cord (Schmidt). In one case recorded by Jakschatsch, there was cystic dilatation of the canal of the cord at the level of the seventh dorsal vertebræ of a calf showing pronounced symptoms of paresis.

**Parasites.** The *Cœnurus cerebralis* frequently penetrates into the lumbar portion of the cord in sheep and cattle, and exceptionally also in the horse, and develops into cysts between the membranes that are elongated in shape and about the size of a finger (so-called lumbar staggers). *Echinococci* and *Cysticerci* occasionally occur (in the pig and dog), and *Echinococci* occasionally develop in the bones themselves (Feuereissen, Goldmann and Stroh). According to Hinrichsen, Brauer, and



Horne, the *Hypoderma lineata* occurs comparatively frequently in the second stage of its development in the epidural fat in cattle (according to Hinrichsen in forty to fifty per cent of slaughtered cattle). Symptoms are produced very rarely because they leave the cord before their development is complete.

**Abscesses** have been observed very rarely in the spinal canal (one case, Raoul). The penetration of pus through the intervertebral spaces from abscesses in the immediate neighborhood of the vertebrae is observed somewhat more frequently. In some cases, pus enters the canal by extension of suppurative processes involving bone as in the case of glanders (Chauvrat, Decoste, Aubury, Boisse, Stanton, Tapken).

**Pathogenesis.** Any of the primary conditions mentioned may lead to a gradual reduction in the size of the vertebral canal, the result being that the cord and nerve roots are subjected to pressure. This occurs earliest in the dorsal region where the canal is comparatively small, and latest in the posterior half of the lumbar portion. Many processes cause a local edema of the spinal cord owing to obstruction of the blood or lymph circulation. Hard growths lying outside the cord hinder its motility and consequently tend to cause tearing or crushing of the cord and nerve roots. Degeneration of the intervertebral discs, or caries of the bone, are likely to lead to excessive curvature of the spinal column at the diseased part, dislocation, or fracture of individual bones (Liénaux), and thus lead to compression of the cord or nerve roots. Finally, chronic inflammation of the cord is set up by the persistent mechanical irritation.

According to Jakob degeneration of the intervertebral discs plays a primary part in the paraplegia which frequently occurs in dachshunds, but in some cases a secondary part through excessive flexion of the spine. Paraplegia is caused far more frequently by stretching and tearing of the peripheral nerves, and particularly at their point of exit through the intervertebral foramina. The correctness of this idea, which is not based upon an analysis of the clinical aspect of the disease, is opposed by the fact that the paraplegia involves all the nerves of the hind parts. This usually indicates supranuclear paralysis and is associated with incontinence of urine and feces.

**Anatomical Changes.** In addition to the lesions of the spine and membranes already mentioned, the cord is reduced to about a third of its normal thickness at the point where the pressure has been applied. The consistence is often reduced, but in rare cases that have been in existence for some time, it may become more dense. The appearance of the surface on cross section is asymmetrical, the shape of the gray cornua and of the inferior fissure being altered. In very exceptional cases there are no visible lesions of the cord, although the animal has shown severe symptoms during life.

Exhaustive histological examinations were carried out by Dexler in cases in which the compression of the cord was due to enchondrosis, enostosis, or neoplasms. At the point where pressure had been exerted and for a certain distance on each



side the nerve fibers were destroyed, the vessel walls were thickened, and their sheaths were infiltrated with round cells. The nerve cells were more or less degenerated, and, in cases that had been in existence for more than a year, the tissue of the cord was invaded by a fibrous tissue that was poor in cells but which contained here and there a few unaltered fibers or groups of nerve cells. In some cases the fibrous tissue contained numerous cavities containing a serous fluid (greatly dilated lymph spaces). In the other parts of the cord there were degenerations of a secondary nature. Above the seat of the compression the posterior columns degenerate as far as the nuclei in the medulla as do also the lateral cerebellar tracts and Gower's tract. The motor tracts and the comma tract of Schultz degenerate in the caudal direction. At the seat of the compression the nerve fibers contained in the nerve trunks originating from the degenerated motor nerve roots may also be in a condition of degeneration.

**Symptoms.** A common symptom is the evidence of pain which sometimes precedes the other symptoms by long periods. The pain may be either constant, in which case the animal holds the spine rigid and somewhat arched, or it may be intermittent and manifest when the animal gets up or lies down, and during movement or jumping, causing the animal to cry out. Owing to the pain, movements are executed with care. In some cases in which the cervical portion of the spine is involved, the animal may be unable to take food from the ground or to graze.

Motor disturbances may be observed in some cases simultaneously with the appearance of symptoms of pain. In other cases they appear later, but they may be observed without any preliminary symptoms indicative of pain. These disturbances, as a rule, first affect the hind quarters irrespective of the seat of the disease, owing to the fact that the muscles of the hind quarters play a greater part in the execution of body movements than those of the fore limbs, and consequently any weakness in them is observed sooner. It is only in exceptional cases that the weakness sets in in all four limbs at once, and it scarcely ever happens that the fore limbs are affected more severely, or exclusively so. As a general rule, the loss of power is bilateral, but in some cases this is not so, and the difference may be so pronounced between the two sides that a superficial examination may lead one to think that the paralysis is unilateral or involves a single limb only. In exceptional cases, if for example only one side of the cord is subjected to pressure, there may be a true unilateral paralysis or monoplegia (Brown-Séquard type). Goldmann records a case in which a pig showed sensory and motor paralysis of one hind leg, owing to an echinococcus in one of the dorsal vertebræ.

In **typical cases**, motor disturbances are observed in the larger animals. The animals rise slowly and with caution, cattle get up like horses, or remain sitting like dogs for a long time. In some cases, they lower themselves on their hind legs while urinating, in the manner frequently observed in puppies (Hamoir and Stenström). In such cases, weakness of the hind quarters is always observable and especially when the animal is turning. Small animals, and especially dogs, are no longer able to jump up or to stand on their hind legs.

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Both large and small animals lie down a lot, are easily fatigued, and during movement it is quite obvious that the limbs are neither lifted nor flexed as much as usual. The latter is especially obvious in the hind limbs, but sometimes also in the fore limbs. In consequence of this abnormality of movement, the hoof wall in the large animals, and the toes in the small are dragged along the ground, especially if the ground be uneven. In dogs the dorsal surface of the toes may be sore. In the later stages, animals walk on the dorsal surface of the phalanges or all the joints knuckle over at the moment when



Fig. 101. Sitting position in spastic paralysis of the hind quarters.

any weight falls on the limb. In still later stages, this symptom is observed while the animal is standing still, or during the passage of urine. After a variable interval, the paralysis progresses to such an extent that the animal can no longer get up or walk. Pressure upon the croup is sufficient to cause them to show signs of falling, and in small animals pressure upon the sacrum causes them to fall powerless to the ground. Finally, in some cases in which there is no paralysis of the fore legs, the animals tend to drag the paralyzed hind quarters after them.

Associated with the gradually progressive symptoms of pa-

ralysis, and the slowly increasing but slight atrophy of the affected muscles, there is in some cases an active rigidity of the muscles in the portion of the body posterior to the seat of compression of the cord. This rigidity is evidenced by a firmer consistence and an increase in the passive resistance offered by the muscles, and also by extension and even crossing of the paralyzed limbs. This is especially obvious when the animal is sitting or lying down (fig. 101). In consequence of the rigidity of the muscles, which in some instances is pronounced while the paresis is still slight, the gait is spastic, the joints are scarcely flexed, and the feet are dragged along the ground. Dogs walk on the tips of their toes (fig. 97, page 682), slip up on smooth surfaces and fall over if they attempt to jump upon anything. In many cases, on the other hand, the feet are lifted backwards and quickly put down again.

In numerous cases that have been examined no actual ataxia has been discovered either by Dexler or Hutyra & Marek. The very variable symptoms are entirely caused by loss of muscle power or by the muscular cramp.

In addition to the pain already mentioned, sensation behind the seat of compression appears to be decreased or even quite lost, but not rarely, and even when there are pronounced symptoms of paralysis, there may be very slight disturbances of sensation or none at all. The insensitive area is not rarely limited anteriorly by a hyperalgesic zone which corresponds with the area supplied by the sensory nerves, the roots of which are subjected to pressure. The pain may be so severe that the animals bite the affected part until they draw blood. If there is tumor-, callus-formation, or luxation, the spine shows a circumscribed swelling, and in many cases of caries there may be a recognizable excrescence. In small animals, there may be evidence of pain if the superior spines be struck or subjected to pressure and even during passive motion. Exceptionally, it is possible to determine pronounced passive mobility of some part of the spine.

Posterior to the seat of compression, the reflexes are exaggerated, but in parts where the compression is somewhat more extensive, there may be a complete absence of reflexes. The frequent erections of the penis which are easily caused by stimulation of the skin of the abdomen, of the prepuce or the penis are the result of the exaggerated reflex irritability.

Abnormalities of function of the bladder and rectum are frequently observed. In view of the fact that the compression is usually situated in the middle of the lumbar portion of the cord, there is, as a rule, retention of urine and feces. In many cases, the animals are unable to pass urine or feces voluntarily. In a greater proportion of cases there is an involuntary discharge of urine and feces at long intervals. This discharge can also be induced by external influences, such as pressure on the wall of the abdomen, or the region of the bladder or perineum. Urination takes place at very short intervals, sometimes



even on extremely slight irritation, and consequently it appears as if the urine were trickling away owing to paralysis of the sphincter, but that this muscle is uninjured is rendered obvious by the fact that urination ceases when the pressure is removed, and also that large quantities of urine collect in the bladder during complete rest. In very rare cases of compression of the lumbar portion of the cord there is observed paralysis of the sphincter of the bladder and dribbling of urine, relaxation of the sphincter of the anus may also be seen. The distribution of the motor and sensory disturbances according to the position of the part of the cord that is subjected to pressure, has been dealt with in connection with contusion of the cord (see page 686).

The occurrence of **atypical cases** is not rare. In some such cases symptoms of paralysis follow disturbances which are inconsiderable, and which possibly have very little local effect. It happens now and then that some intraspinal growth for a long time causes no obvious symptoms and is then the cause of the sudden onset of symptoms. In both cases the deviation from the usual course of events is caused by crushing or stretching of the cord or some of the nerve roots through some sudden movement or excessive flexion of the spine, thus causing either a unilateral traumatic lesion or probably some interruption of conductivity due to a local disturbance of circulation.

**Course.** Corresponding to its nature the condition runs a chronic course. In typical cases the disturbances increase until there is complete paralysis which may be gradual, or there may be times at which the disease is at a standstill, or possibly there may be some improvement. In other cases some slight disturbance is followed by severe or complete paralysis, or there may be severe symptoms of paralysis which are not preceded by any obvious disturbance. In cases of this kind in small animals, the condition may abate for a certain length of time or even completely disappear, but as a rule, after some weeks, it makes its appearance again, and this may occur repeatedly. In exceptional cases the improvement is maintained so long that from a clinical point of view the animal may be said to have recovered. In the large animals such improvements are not observed because of the development of bedsores or of hypostatic congestion of the lungs.

In the majority of cases the disease terminates fatally, and usually owing to complications. Bedsores develop quickly in the larger animals, but are not absent in the later stages in small animals. As a rule these lead to a general infection. The rapid course of the disease in the larger animals is referable to this and also to hypostatic congestion of the lungs. In comparatively rare cases there is catarrhal inflammation of the urinary tract.

**Diagnosis.** Diagnosis is associated with little or no difficulty in cases in which there are the following symptoms: very slowly progressive paraparesis and para-anesthesia, local swelling and pain of the vertebral column, evidence of spontaneous pain, a girdle-like area that is hyperesthetic, and disturbances of micturition and defecation. An error of diagnosis is far more likely to be made in those cases in which the paralysis appears to set in or actually sets in suddenly. The presence of more or less pronounced atrophy of the muscles and spastic movements of the paralyzed portion of the body lead one to suspect that certain motor disturbances have been in existence for some time. Possibly also there is a history of the occurrence of rheumatic pains and stiffness of gait.

The points that are of importance in the differential diagnosis of compression of the cord, ossifying meningitis, contusion of the cord, and myelitis, have already been dealt with under these diseases. Muscular rheumatism with which the disease may be confused owing to the evidence of pain, is easily distinguished by the facts that there is no loss of muscular power, rigidity of the muscles is absent, the sensibility of the skin is increased and the reflexes are normal. In the horse, thrombosis of the branches of the posterior aorta might be confused with the disease, but this is distinguished by the fact that the paralysis which is observed during movement rapidly disappears when the animal is left at rest, and rectal examination enables one to establish the diagnosis.

The seat of the compression can only be determined accurately in cases in which there is some distortion of the body; there is local pain of the vertebræ, a hyperesthetic zone can be demonstrated, the extent of the paralysis and loss of sensation, the condition of the reflexes, and the functions of the bladder and rectum also afford information. In other cases one can do no more than determine the approximate seat of the lesion, and in some cases this amounts only to deciding that it is situated in the cervical, dorsal, lumbar or sacral sections of the spine.

The cause of the compression can in some rare cases be accurately determined. In this connection, the following points must be taken into consideration. In the ox and pig, tuberculosis is the commonest cause of compression of the cord, in the sheep cœnurosis, in the dog disease of the vertebral joints. Tuberculosis of the internal organs strongly suggests the possibility that this is the cause of the trouble, the presence of gid in a flock suggests the parasitic nature of the condition, the presence of neoplasms in other parts of the body, and especially in the neighborhood of the vertebral column is more suggestive of a growth in the canal being the cause of a reduction in its lumen. If the symptoms of compression set in during the course of a suppurative disease the probability is that the cause is an abscess.

**Prognosis.** This, as a general rule, is unfavorable, and especially in large animals which are already unfit for work, owing to somewhat severe motor disturbances, and in which dangerous complications are likely to occur early. In the small animals a complete recovery is not to be expected. Not rarely the animals improve and the symptoms of paralysis disappear, but the disappearance is only temporary, and, inasmuch as the possibility of a regeneration is not to be expected, a recovery in the clinical sense cannot be hoped for. On the grounds of the symptoms presented one cannot say whether there is likely to be an improvement later or not, for this purpose the animal must be under observation for a week or two, during which time in favorable cases some improvement will be noticeable.

**Treatment.** No satisfactory treatment can be advised in the case of the large animals. They should be slaughtered as soon as possible. In the small animals the extirpation of neoplasms may be attempted, but in the majority of cases radical treatment cannot be adopted owing to the impossibility of exactly localizing the disease. In such cases there is not rarely some improvement and even a complete disappearance of symptoms if all movement be avoided and the animals kept in a condition of complete rest. The animal's position should be changed from time to time, and the urine and feces should be removed. If the animals can move moderately well, slight movements and massage of the muscles may be useful. Some authors have observed improvement follow the injection of eserine and pilocarpine, but such improvement might have happened without any such treatment.

**Literature.** Barrier, Bull., 1906, 283.—Dexler, Ö. Z. f. Vk., 1896, VII, 1; *Ergeb. d. Path.*, 1896, III, 2, Abt., 516 (Lit.); 1900, VII, 479 (Lit.); *Nervenkrkh. d. Pferdes*, 1899, 87 (Lit.).—Feuereissen, Z. f. Flhyg., 1905, XV, 86.—Fröhner, Monh., 1899, X, 123.—George & John, S. B., 1885, 40.—Goldmann, Z. f. Flhyg., 1907, XVIII, 35.—Hamoir, Ann., 1904, 627; 1906, 332.—Haugmeier, Rep., 1853, 112.—Hink, D. t. W., 1899, 4.—Hinrichsen, A. f. Tk., 1882, XIV, 219.—Jakob, M. t. W., 1910, 305.—Jakschatsch, B. t. W., 1899, 455.—Kammermann, Schw., A. f. Tk., 1888, XXX, 205.—Nocard, Bull., 1885, 80.—Petit, Rec., 1906, 470.—Poes, Ann., 1902, 89.—Poulin, *ibid.*, 1906, 687.—Rubay & Navez, *ibid.*, 1902, 629.—Schmidt, A. f. Tk., 1889, XV, 295.—Stenström, Z. f. Tm., 1906, X, 113.—Tapken, D. t. W., 1905, 482.—Teetz, Z. f. Flhyg., 1905, XV, 60.—Wilson, J. of comp. Path., 1904, 332.

## 7. Syringomyelia.

This name is applied in human medicine to a condition in which there is an increase, and later a destruction of the neuroglia tissue with the production of cavities, the walls of which are formed of neuroglia tissue. The cavities that are formed by dilatation of the lymph spaces, or to softening of the tissues, observed in cases of myelitis, due to pressure and in other forms of myelitis, are not included under the term.

There are only two records of the occurrence of syringomyelia in



the lower animals. One is recorded by Roger as occurring in the guinea pig; and the other in the dog by Liénaux. These two cases, and the histological examinations made by Liénaux do not definitely settle the question whether the condition found in these animals exactly corresponds with the condition found in man, as Liénaux supposed in his case. In this case symptoms of posterior paraparesis gradually developed in a two-year-old Newfoundland. After four months, there was loss of sensation in the hind quarters, and later there were clonic contractions of the digastric muscle which caused occasional opening of the mouth. In the sixth month, the gait was unsteady, the croup was sunken owing to excessive flexion of the joints of the legs; and, owing to paralysis of the muscles on the left side, there was lateral curvature of the spine the convex side of the curve being on the left. By this time the animal only got up to feed or when told to do so, but soon lay down again powerless. From the scapular region backwards the left half of the body was completely insensitive; while there was marked hyperesthesia of the right side. The patellar reflex which, at the outset was normal, was exaggerated on both sides. Later the area of anesthesia spread to the neck. There was no disturbance of micturition or defecation.

At the postmortem, there was found a cavity extending the whole length of the cord, the walls of which were covered with ependyma, and which in the dorsal and lumbar portions of the cord, communicated with small cavities scattered through the gray matter. When cut into, a clear serous fluid escaped from this cavity. Microscopic examination showed atrophy of the nerve cells, increase of the neuroglia cells, slight unimportant perivascular infiltrations, and a secondary degeneration extending up to the medulla.

**Literature.** Liénaux, *Ann.*, 1897, 486.—Lefébure, *Rec.*, 1906, 516.

### SECTION III.

## DISEASES OF THE PERIPHERAL NERVOUS SYSTEM.

### General Etiology and Symptomatology of the Diseases of the Peripheral Nerves.

A variety of causes are responsible for diseased conditions of the peripheral nerves.

**Traumatic injuries** are frequent causes of such conditions. In the first place a superficial nerve may be crushed between the underlying bone and some hard object, or more rarely between a muscle in a condition of powerful contraction and the bone. In a number of cases nerves are injured by blows, or penetrating wounds, but tearing of a nerve owing to sudden excessive extension of a limb is observed only very occasionally. Solution of continuity of the nerves is rarely met with in cases of wounds of the surrounding soft tissues or fractures of bones.

**Compression of nerves** may result from the development of neoplasms either around the nerve, in its immediate neighborhood, or even in the nerve itself. Neuromata occur almost exclusively, but with moderate frequency in the ox. It is only exceptionally, however, that they give rise to any symptoms (Zietschmann, S. B. 1900, 234; Detroye, Rev. Vét. 1907, 408). As a rule, several nerve trunks of the head, trunk and limbs are involved simultaneously, and they also occur on connection with the fibers and ganglia of the sympathetic. They are composed of fibrous and gelatinous tissue and arise in connection with the interstitial tissue of the nerves, the nerve elements remaining uninjured. In some cases, amputational neuromata develop after neurotomy has been performed, and they are the cause of severe pain.

Nerves may suffer compression owing to the existence in their neighborhood of enlarged and indurated lymphatic glands, effusions of blood, abscesses, foreign bodies, parasites (larvæ of *Hypoderma*), dislocations of bones and calluses resulting from fractures of bones.

**Neuritis.** Recent investigations have furnished evidence that neuritis is by no means an uncommon disease in the domesticated animals. The explanation of the scanty records of the occurrence of neuritis in the older literature is that in those times it was the central nervous system that was principally examined in cases in which there were nervous disturbances, the peripheral nervous system receiving no attention at all or a mere naked-eye inspection. Consequently, the inflammatory changes in the nerves which are only recognizable on microscopic examination passed unnoticed.

There is no doubt that cases occur in which the neuritis is due to chilling, but in what manner the effect is produced is not accurately known. There is but little information as to the alterations that occur in nerves as a result of cold, and it can only be maintained with a certain amount of probability that they are of an inflammatory nature. In the vast majority of cases neuritis is due to an infection. The inflammation in such cases is set up by microorganisms or, as appears to be more commonly the case, by their toxins. At the present time, the causes of dourine, strangles, and influenza are known to be capable of setting up neuritis. The results of more recent investigations have shown that certain poisons may cause neuritis, provided the animals survive sufficiently long, as in the case of chronic poisoning, or after recovery from a not very severe poisoning. Thomassen furnished experimental proof that lead possesses the power of causing neuritis in certain nerves. Up to the present no evidence has been adduced with regard to other poisons, but it appears to be probable that the same holds good for mercury and arsenic. Many vegetable poisons of known and unknown composition appear to be capable of setting up neuritis. Satisfactory and conclusive experiments and observations have been made regarding the neuritis caused by polished rice. Mechanical influences (stretching, blows, pressure, etc.) set up chronic inflammation in the connective tissue of nerve trunks, especially if they are slight and are in action for a long time, or affect one nerve repeatedly. Finally, some cases may result from the extension of inflammatory processes that are going on in the neighborhood of the nerve, for example, pleurisy may involve the recurrent nerve, the glosso-pharyngeal nerve, may be affected in pharyngitis or disease of the pharyngeal organs, and severe inflammation of the lymphatic glands may involve any nerve that is near.

Lesions that are visible to the naked eye are found in only a small proportion of cases, and then only in cases in which the inflammation has led to the production of a large amount of connective tissue, pronounced degeneration, or atrophy. The gelatinous appearance of the perineural connective tissue in animals that are greatly emaciated must not be considered as an inflammatory condition, since the gelatinous infiltration is



simply the result of the removal of a considerable quantity of fat. Definite results can be obtained only by microscopic examination which reveals, in acute cases, dilatation of the vessels, cellular infiltrations, small hemorrhages in many cases, in chronic forms an increase in the amount of connective tissue, and in both cases degeneration and disappearance of the nerve fibers extending in the peripheral direction from the seat of the disease. If the seat of the inflammation is on the central side of the spinal ganglia or the ganglia of the cranial nerves, the degeneration is found in the sensory roots of the spinal or cerebral nerves and in the posterior columns of the cord or in the sensory root of the trigeminal nerve. As a rule the lesions do not involve the whole cross-section of the nerve trunk to the same extent.

If the inflammation starts in the connective tissue surrounding the nerve it is known as perineuritis, but if it has its starting point in the intraneural interstitium it is called interstitial neuritis. The term parenchymatous neuritis is reserved for those cases in which the primary lesion is a degeneration of the nerve fiber.

**Diseases of the central nervous system and its membranes** frequently cause paralysis and other functional disturbances of the peripheral nerves, but, strictly speaking, these do not belong to the diseases now under consideration. On practical grounds it appears to be advisable, however, to refer to them while the subject of the diseases of the peripheral nervous system is under discussion.

Diseases of this kind involving the purely motor nerves abolish the conductivity of the affected nerves, either entirely or in part, either through the entire cross-section of the nerve or in a part only. This results in an infranuclear (peripheral) paralysis of the muscles supplied by the nerves in question.

In mixed nerves the conductivity is preserved for a time, and not rarely till the time of death in the sensory nerve fibers which are apparently more resistant, provided the nerve be not completely severed or crushed. In view of the fact that the sensory nerves are stimulated more easily by various influences, disease of a portion of a mixed nerve, as a rule, leads to pain through the whole length of the nerve and throughout the area supplied by it. The contractions and spasms of the muscles concerned and the exaggeration of the reflex irritability which is observed in rare cases are due to stimulation of the sensory fibers. The reflexes persist so long as the conductivity of the motor nerves is not destroyed. As the motor nerves which are very easily injured lose their conductivity right from the start or very soon after, muscular spasms and exaggeration of the tendon reflexes are very rarely observed in diseases of the peripheral nerves, except in disease of the membranes, in which the entirely different mode of development

often produces the opposite effect. Infranuclear paralysis is often associated with the loss of conductivity in the motor fibers in a mixed nerve. Sensation persists in the paralyzed area, and it may be either exaggerated, but it is diminished or completely lost if the conductivity of the sensory nerves has been destroyed by some very severe inflammation.

### 1. Paralysis of the Nerves of the Eye.

**Etiology.** Paralysis of the III, IV and VI pairs of cranial nerves is generally due to some intracranial disease (meningitis, concussion of the brain, encephalitis, tumors), and is of fairly frequent occurrence under such circumstances. (In a case recorded by Zschokke, the abductor nerve was subjected to pressure by an intracranial angioma.) There are no cases on record of extracranial disease of these nerves, but injuries might occur through fractures of bone in their immediate neighborhood, or through the presence of exostoses or tumors in the depth of the orbit.

In cases of torsion of the eyeball recorded in horses by Ballangée and Bayer, Clerchet, Fayet & Nicolas, the cause was not discovered.

**Symptoms.** Paralysis of the oculo-motor nerve is observed most frequently, the condition being more rare in the abductor and trochlear nerves. In cases of paralysis of the oculo-motor the upper eyelid is dropped (ptosis), the animal is unable to open the eye, but is able to lift the upper lid to a certain extent by powerful contraction of the corrugator supercilii, which is supplied by the facial nerve. The bulb of the eye is directed outwards and its outer half somewhat upwards (strabismus divergens). The pupil is dilated and does not react to light. On pressure the eye is not retracted into the orbit and the membrana nictitans is consequently not seen.

Paralysis of the external rectus muscle, which is supplied by the abducens nerve, causes the eyeball to turn inwards (strabismus convergens), while paralysis of the trochlear nerve, which supplies the superior oblique muscle, causes the eye to revolve with its outer part downwards.

Dexler has repeatedly observed exophthalmus in cattle, also convergent squinting, and functional disturbances of the muscles of the eyes associated with secondary catarrh of the conjunctiva. The cause was in all probability an increase in the intraorbital fat.

**Prognosis.** Improvement is scarcely to be hoped for, but it depends upon the nature of the primary disease. Exceptions to this rule are many cases of concussion of the brain or rotation of the eyeball without other disturbances (Ballangée).

**Treatment.** In view of the fact that the origin of the disease is as a rule central, treatment should be along the lines laid down for diseases of the brain or its membranes.

**Literature.** Ballangée, A. f. Tk., 1906, XXXII, 103.—Clerchet, Fayet and Nicolas, Bull., 1909, 490.—Zschokke, Schw. A., 1885, XXVII, 174.

## 2. Paralysis of the Trigeminal Nerve.

**Occurrence.** This paralysis has up to the present been observed in the horse and dog, and Reuschel claims to have seen it in a cow. (Hintze found degenerative atrophy of the infraorbital nerve in a pig affected with rickets of the jaw.) In the dog, the disease is not rarely observed, even if cases of rabies, in which it is common, and cases of distemper, in which it is infrequent, are excluded.

**Etiology.** The principal causes of paralysis of the trigeminal nerve in the dog are rabies and distemper, but among other causes may be mentioned inflammation of the brain in the neighborhood of the pons (Mathis), concussion of the brain (Hutyra & Marek), the development of a neoplasm around the intracranial root (Gratia, and Cadéac & Roquet, a gliosarcoma and an endothelioma) and, under certain circumstances, squeezing of the motor branch through powerful contraction of the muscles of mastication (laying hold of some heavy object, biting a hard bone, etc.).

The crushing of the motor branch between the masseter and temporal muscles is explained by supposing that the nerves mentioned are pressed against the condyloid process during active contraction of the temporal muscle. This is particularly the case when a dog forcibly closes its widely opened mouth or is holding a heavy object in its mouth. A consideration of the anatomy of the part is sufficient to convince one that a crushing of the motor branch only, and one that is as a rule bilateral, is quite possible, and it is not necessary to suppose that the cause is either rheumatism or some intoxication, as Cadot and Almy assert. The sudden onset of the disease contraindicates this.

The disease is met with only very occasionally in the horse. In a case recorded by Lydtin, the cause of the paralysis was a fibrosarcoma in the immediate neighborhood of the Gasserian ganglion, and a growth of the same nature close to the temporo-maxillary joint is recorded as the cause by Tempel. Meyer, in one case ascribes the paralysis to the presence of an angioma in the cranium, and neuritis is said to be the cause in cases mentioned by Röhl, Lüpke, Dexler and Marek. Sometimes trigeminal paralysis appears to be due to concussion of the brain (Berton, Pr. Mil. Vb.).

**Symptoms.** In cases of paralysis of all three branches of the trigeminal nerve both, disturbance of mastication and loss of sensibility are observed. If the first branch (ophthalmic



branch) be alone affected the skin over the forehead up to the level of the ears (fig. 102), the eyelids, the nasal mucous membrane, the surface of the eyeball and the cornea are insensitive. Pressure on the cornea does not cause closure of the lids nor retraction of the eye. Particles of dust and dirt settling on the surface of the eye are not perceived and consequently not removed. As a result of this the cornea becomes cloudy and soon ulcerates, thus allowing the inflammation to spread to deeper parts of the eye (*ophthalmia neuroparalytica*).

If the second branch be paralyzed (the superior and inferior maxillary branches) there is loss of sensation of the skin of the face, of the dorsum of the nose, the cheeks and lips and the mucous membrane of the tongue on the diseased side (fig. 102). The tongue is injured during mastication without the animal feeling it. The upper lip on the diseased side is drawn towards the opposite side owing to loss of muscular tone, while the insensitive half of the lower lip moves slowly. Simultaneous paralysis of the vaso-motor nerves may cause intense reddening of the mucous membranes. In cases of paralysis caused by compression the anesthesia is always preceded by hyperesthesia.

Disease of the lower branch causes also paralysis of the muscles of mastication (masseter, temporal, internal and external pterygoids). The animal masticates with the sound side only, the teeth of the opposite side scarcely coming into contact with each other. One can easily convince oneself of this by allowing the animal to chew some hard object. The lower row of incisors is pulled away from the paralyzed side and a narrow space is left between the upper and lower rows. The teeth on the paralyzed side do not wear properly and particles of food collect between the cheek and the teeth owing to the insensitiveness of the mucous membrane. If the hand be placed on the temporal region or on the masseters during mastication it can be noticed clearly that while the muscles on the sound side contract, those on the opposite side remain quiescent. In time the paralyzed muscles undergo atrophy.

In dogs paralysis of the trigeminal nerve resulting from crushing by the contraction of the muscles of mastication tends to be bilateral and involves the motor branch only. The mouth is held open and the lower jaw is dropped, but it is easily raised. The tongue is dry and hangs out of the mouth, there is a flow of saliva (fig. 102), food and water cannot be taken, but food



Fig. 102. Paralysis of the trigeminal nerve due to contusion of the brain. The dark line indicates the upper and posterior line of the anesthetic area.

placed upon the tongue is gulped down with some effort. In some cases rabies, and exceptionally distemper, may be accompanied by bilateral paralysis.

Paralysis due to diseases of the brain is in many cases only partial, the loss of sensibility is often limited to one or the other branch of the nerve, while a partial disease of the long trigeminal nucleus or the root appears to be quite possible.

In many cases facial paralysis follows paralysis of the trigeminal nerve (Dexler, Tempel and Marek), or there may be symptoms of bulbar paralysis. In a case of paralysis caused by a subparotid abscess Dupas observed simultaneously hyperidrosis and hyperthermia of the head and part of the neck, and hyperesthesia of the larynx.

**Course.** In the dog paralysis resulting from a blow on the peripheral portion of the nerve may be cured within one or two weeks or in some cases later if the animal be fed artificially in the meanwhile. In some cases, however, the paralysis is persistent, in which case there is pronounced atrophy of the muscles of mastication. Paralysis due to concussion of the brain is in some cases curable, but paralysis due to other causes does not tend to disappear.

**Diagnosis.** There is no difficulty about recognizing paralysis of the motor and sensory branches, but the discovery of the cause is sometimes associated with some difficulty. Differential diagnosis of the paralysis observed in rabies from the other forms of bilateral paralysis is easy when the change in the habits of the animal, its appearance, the disturbance of appetite and swallowing and the weakness of the hind quarters are taken into consideration.

**Treatment.** In cases of paralysis of the jaws resulting from concussion the principal thing is to provide the animal with nourishment artificially. This may be done either with a stomach tube or by simply placing pulpy or somewhat more solid food on the root of the tongue. Further treatment appears to be superfluous except massage and the application of electricity. Similar treatment may be adopted in cases resulting from concussion of the brain. Treatment in other cases is useless.

**Literature.** Berton, *Rev. Gén.*, 1903, I, 455.—Cadéac and Roquet, *J. Vét.*, 1908, 65.—Dexler, *Nervengerkrh. d. Pferde*, 1899, 26 (Lit.).—Franz, *B. t. W.*, 1903, 40.—Meyer, *Monh.*, 1904, XV, 60.—*Pr. Mil. Vb.*, 1902, 92.—Regenbogen, *B. t. W.*, 1907, 325.—Reuschel, *W. f. Tk.*, 1907, 706.

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**Spasms of the Muscles Supplied by the Trigeminal.** These are observed in cases of tetanus (trismus) and they also appear to be somewhat frequent in cases of distemper, in which cases there are more or less rhythmic contractions. The contractions occur either on one or both sides causing the teeth to be brought together in such a way as to produce a distinct cracking sound.

Acute cerebral meningitis may, in some cases, give rise to tonic or clonic contractions of the muscles supplied by the trigemini.

Tonic-clonic spasms have been observed in the area supplied by the trigemini in a horse showing symptoms of sleepy staggers (Hutyra & Marek). The lower jaw was pressed spasmodically against the upper, the angle of the lip and the upper lip on both sides were drawn upwards, and the lower lip downwards, giving the horse an appearance resembling a dog gnashing its teeth. The muscles of mastication showed more or less persistent contractions. The attacks occurred in a remarkable way during feeding, every time the animal opened its mouth a little widely. There were no detectable disturbances of sensibility or other abnormalities to be discovered in the mouth.

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**Neuralgia of the Trigemini** (Tic douloureux). Bielefeld, Liautard, Strebel and Williams claim to have observed the disease. Williams observed it in horses of very active temperament, and more frequently during movement than rest. There was restless tossing of the head and rubbing of the lips against any object at hand. In three cases resection of the infra-orbital nerve caused disappearance of the symptoms, while in one case there was only improvement. Bielefeld observed increased sensitiveness and hyperidrosis in the area supplied by the nerves.—(Bielefeld, *Pr. Mt.*, 1856-57, 103.—Liautard, *Bull.*, 1903, 40.)

### 3. Paralysis of the Facial Nerves.

**Occurrence.** Paralysis of the facial nerves occurs with moderate frequency in the horse, but only rarely in the other species.

**Etiology.** Anatomical considerations and the general conditions of their employment suggest that traumatism is the principal cause of facial paralysis in horses, the point where the nerves turn the posterior border of the jaw being exposed to various injuries. In many cases cold seems to play a part, especially when the animal is hot and stands facing or sideways to a cold wind, or when in winter an animal stands opposite an open window or door in the stables.

In rare cases the paralysis may be caused by inflammatory infiltration of the pharyngeal and parotid regions, the growth of a neoplasm involving the ligaments of the temporo-maxillary articulation (fibrosarcoma, Tempel), a subarticular melanoma, a dental cyst (Deghilage), or an abscess in the long cornu of the hyoid bone (Jewtichilew). Sometimes neuritis is the cause. Dexler found extensive cellular infiltration and degeneration of the nerve fibers in both facial nerves in the neighborhood of geniculate ganglion. The facial paralysis which appears to be common in dourine is due to a neuritis, and the same holds good in the case of paralysis seen in some cases of influenza. In many cases paralysis of the facial nerves is due to disease in distant organs (wounds, laminitis, cellulitis, colic), and according to Dieckerhoff the cause in such cases is a septic intoxication. In view of the fact that the nerve is exposed to injury at the point where it turns round the jaw the cause of the



paralysis in these cases is probably an injury due to the animal lying down for a long time or rolling, and especially in unsuitable stables. Other causes are: inflammation of the middle ear, caries of the petrous temporal bone, neoplasms in the base of the brain, inflammation or contusion of the pons or anterior portion of the medulla, thrombosis of the inferior cerebellar artery (Vosshage), etc.

In **cattle** paralysis of the facial nerves sometimes results from inflammation of the middle ear (Leblanc), epidermal collections in the passage of the ear, actinomycosis in the parotid region (Fuchs), tuberculosis of the brain (Fuchs). It is only exceptionally that it is caused by mechanical injury, because the facial nerve is protected by the horns and it is not exposed to injury for half its length. Paralysis of the facial nerve following parturient paresis is obviously of traumatic origin.

Cases resulting from chill have been recorded in the **dog** (Trossinow observed it in a dog after being brought out of cold water), neoplasms in the parotid region (Gratia, sarcoma), more frequently from inflammation of the middle and internal ear, caries or tuberculosis of the temporal bone (Montfallet), and finally encephalitis due to distemper.

**Symptoms.** In cases in which all the branches of the nerve are paralyzed the following symptoms are observed. Owing to paralysis of the muscles of the ear that organ occupies a horizontal position or even hangs down on the paralyzed side. In dogs with naturally hanging ears it hangs over backwards or is quite normal in position, but does not show movements corresponding to the ear of the opposite side (fig. 103). Paralysis of the orbicularis palpebrarum abolishes the power of closing the eye and as a result the eye remains open but the palpebral fissure appears narrower than on the opposite side, or the upper lid hangs down somewhat because the corrugator supercilii which assists in the elevation of the lid is also paralyzed. The sudden meeting of some obstacle does not result in closure of the eye, but if the cornea or conjunctiva be touched the membrana nictitans is pushed forwards, the eyeball is turned inwards and upwards showing that the inability to close the eye results from paralysis of the orbicular muscle and not of the trigeminus. In spite of the absence of power to close the eye there is no tendency to the development of severe inflammatory conditions of the cornea owing to the movements of the eyeball and of the membrana nictitans. There may be slight turbidity and even small superficial losses of tissue in cases of complete paralysis. There is often lachrymation which results in the loss of hair below the inner angle of the eye if it persists for long.

In contrast to what is seen in horses and cattle, the hanging of the upper lid does not appear to be constant in the dog. In a case recorded by Müller the palpebral fissure on the paralyzed side was wider than normal. As a result of experimental section of the auriculo-palpebral nerve in a dog Marek observed marked

drooping of the upper lid so long as the upper lids were not elevated to the maximum, but when this occurred the opening on the sound side was wider. In another dog affected with facial paralysis the palpebral fissure appeared wider only while the dog remained quiet, and this as a result of marked dropping of the lower lid.

In the horse the ala of the nose is relaxed and sunken against the septum. The nostril is elongated and narrow and appears to be lower in position than that of the opposite side (fig. 103).



Fig. 103. Total paralysis of the left facial nerve.

During inspiration the wing of the nostril is forcibly drawn inwards, thus narrowing the opening still more. In the other species only absence of movement of the nostrils is observed during respiration or smelling at objects.

In the horse the upper lid appears to be drawn to the sound side to a marked extent. The angle of the mouth on the sound side is higher and that half of the mouth is a little open owing to the dropping of the lower lip, exposing the edges of the teeth

of the lower jaw (figs. 103 and 104). Pricking the paralyzed lower lip causes no contraction, but the animal moves its head because it feels the prick.

In the early stages the tongue frequently hangs out of the mouth, but later assumes its normal position and is capable of normal movements. Owing to paralysis of the cheeks, particles of food remain between the cheeks and the molar teeth and the mucous membrane is sometimes injured. During drinking the head is sometimes held obliquely, the paralyzed side being the lower.

In cases of bilateral paralysis all the abnormalities are present saving the lateral displacement of the lips. In the horse there is great difficulty of respiration owing to narrowing of both nostrils and if the animal be made to move fast this may amount to suffocation (Möller). In cases of bilateral paralysis of the lips there is often difficulty in the prehension of food;

horses bury their mouths deeply in food or water and drink in a manner resembling that seen in pigs. When drinking out of shallow vessels they make lapping movements with the tongue. In cases of severe bilateral paralysis a portion of the water taken in escapes through the nose owing to paralysis of the soft palate.

In mild cases the mechanical irritability of the nerve is either normal or only slightly altered, whereas in severe cases it is soon completely lost.

Paralysis of the facial nerve arising from disease of the brain or its membranes may be associated with paralysis of other cranial nerves and not rarely with hemiplegia of the opposite side (hemiplegia alternans).

Dupas observed hyperidrosis and hyperthermia of the same side of the head and the upper part of the neck, and hyperesthesia of the larynx in a case of paralysis caused by a subparotid abscess (see page 720).

The symptoms described refer to paralysis due to lesions of the nerve trunk, the root or the nucleus. Nothing definite is known regarding paralysis of the facial nerve due to interruption of the central motor path leading to the nucleus of the nerve (supra-nuclear paralysis). Such paralysis may occur in cases of hemiplegia of supra-pontine origin and on the same side. The symptoms that would be expected in such cases would be like those observed in the human subject; persistence and even exaggeration of the reflex irritability, unaltered excitability to mechanical stimuli, the absence of degeneration reaction, and finally, pronounced abnormalities of the lower facial branches.

**Course.** The cases of paralysis due to injury or cold, which, as a rule, are unilateral and limited to the lower branch, recover



Fig. 104. Paralysis of the left facial nerve in a dog associated with twisting of the head owing to simultaneous internal ear disease. The left eye cannot be closed and the left halves of the lips hang down.



in many cases within a few days, but in some cases only after several weeks. It occurs only rarely that a certain amount of weakness of the muscles of the face persists when the paralysis has persisted for a long time. If the seat of the disease is in the temporal bone or the cranial cavity, the prognosis is, as a rule, unfavorable. The same holds good in cases of bilateral paralysis owing to the difficulty of respiration and of taking nourishment.

**Diagnosis.** The symptoms of facial paralysis are so striking and characteristic that it is only the localization and determination of the cause that presents any difficulty. Paralysis due to a lesion of the facial trunk involves all three branches. If the lesion is situated outside the stylo-mastoid foramen there is no paralysis of the muscles of the ear, because the nerves supplying these muscles separate from the main nerve immediately outside this foramen and consequently escape injury. On the other hand, even in neuritis of the trunk, all the branches are not always involved. The nearer the lesion is to the inferior maxillary branch the less likelihood there is of the auriculo-palpebral branch being injured. The orbicular muscle and the corrugator escape if the conductivity of the nerve is destroyed at or near the seat of injury. This is usually the case in the horse. If the nerve be diseased within the temporal bone there may be pain on pressure in this region and loss of hearing. It is also possible that if the nerve be injured in horses near the auditory meatus, the geniculate ganglion or somewhat deeper, there may be regurgitation. If the intracranial portion be diseased there may be functional disturbances of neighboring nerves, the auditory and abducens (deafness and squinting of the eye on the diseased side), and there may even be hemiplegia. In these cases all the branches are, as a rule, involved, but if the nucleus be diseased individual branches may be more or less free. Infranuclear paralysis involving exclusively the lips, nose and cheeks, is almost without exception due to a lesion at the geniculate portion, if no other nervous symptoms are present.

Horses in poor condition or old horses frequently allow the lower lip to hang, but a prick with a needle always causes a contraction. In cases of facial paralysis this movement is absent because the paralysis is almost always of infra-nuclear origin and there is consequently a loss of reflex irritability.

**Treatment.** Paralysis due to an injury or to cold tends to disappear without any special treatment, especially if the animal be turned out. It appears to be probable that auto-massage plays some part in the cure. Systematic massage of the muscles of the face and ear and the application of electricity must be borne in mind. In cases that are due to abscess-formation or the growth of neoplasms surgical interference may have the desired result.

Soft food should be given and particles remaining between

the teeth and the cheeks should be removed. If the usefulness of the animal is impaired owing to bilateral narrowing of the nostrils the collapse of the paralyzed nostrils may be prevented by sutures or tracheotomy may afford relief.

**Literature.** Cadéac, J. Vét., 1902, 526.—Dexler, Monh., 1896, VII, 193; Nervenkrkh. d. Pferdes, 1899, 27 (Lit.); Ergebn. d. Path., 1900, VII, 458.—Dupas, Bull., 1904, 527.—Dutrey, Ann., 1906, 339.—Müller, S. B., 1900, 261.—Pr. Mil. Vb., 1899, 97.—Pesadory, Clin. Vet., 1906, 543.—Reinshagen, Pr. Vb., 1904, II, 62.—Vosshage, D. t. W., 1902, 483.

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**Spasm of the Facial Nerve.** This occurs with moderate frequency in dogs as a result of encephalitis, due to distemper, the cause being inflammation in the neighborhood of the nucleus of the nerve. In a case carefully investigated by Cadiot, Gilbert and Roger, the contractions remained unaltered after destruction of the paths leading to the nucleus of the facial nerve, but disappeared after destruction of that nucleus. The condition has also been observed in cases of meningitis and other forms of encephalitis, and apparently also in cases of simple neurosis. In all these cases, there are clonic spasms of the muscles supplied by the nerve, while in tetanus there are generally tonic spasms.

#### 4. Paralysis of the Auditory Nerve.

**Etiology.** Paralysis of the **cochlear nerve**, the true organ of hearing, is a congenital defect in albinotic animals and is due to a defective condition of the spiral ganglion with resulting degeneration of the organ of Corti (Alexander, Tandler, Beyer). Paralysis of the nerve may also be caused by inflammatory changes in the internal ear (Jakob), or intracranial disease in the neighborhood of the medulla oblongata.

**Paralysis of the vestibular nerve** is frequently observed in dogs, pigs, rabbits and birds. It is often caused by inflammation of the middle and internal ear as in swine plague (Bergmann), in contagious rhinitis, inflammation of the nose caused by coccidia, and in chicken pest. The paralysis may also be caused by inflammation or caries of the petrous temporal bone. Other causes are: concussion of the brain (see page 623), and hemorrhages of the internal ear caused in a similar manner. Other diseases of the medulla or the cerebellum may cause interruption in the conductivity of the vestibular nerve. In pigeons it is observed in cases of contagious meningitis (megrimms).

**Symptoms.** Bilateral disease of the **cochlear nerve** is easily recognized owing to complete deafness, but if the disease be unilateral it generally escapes the owner's notice and is very difficult to demonstrate.

Unilateral paralysis of the vestibular nerve is evidenced in all species by the head being held in an oblique position, the dis-

eased side being held lower (see figs. 104 and 105), but the lower part of the head is generally turned back towards the sound side. This turning of the head may be slight or  $45^{\circ}$  to  $75^{\circ}$ ; but in birds it may be as much as  $180^{\circ}$  or more, so that the roof of the cranium is in many cases in contact with the ground (see fig. 105). Rolling movements are common in the small animals, but quite exceptional in the large, the rolling always being towards the diseased side. Animals sometimes take up strained positions. The abnormal position of the head causes the animals, if they try to move at all, to walk in circles towards the sound side, but sometimes the movements are in the opposite direction or at any rate they cannot walk in a straight line. There is frequently horizontal nystagmus towards the sound



Fig. 105. Paralysis of the vestibular nerve in a fowl. Twisting of the head more than  $180^{\circ}$ .

side with conjugate position of the eyes (see page 593). There may be difficulty in taking food and in birds it is commonly quite impossible.

If the disease is bilateral it closely resembles cerebellar ataxia, only the symptoms are limited to the head and neck.

**Course.** While paralysis due to injury generally disappears completely within a few days to a week, other diseases of the auditory nerve are, as a rule, persistent, depending upon the nature of the primary disease.

**Treatment.** In traumatic cases quiet, and artificial feeding if necessary, are sufficient. If there is coincident disease of



the ear local treatment or surgical interference are indicated. In the so-called megrims of the pigeon an attempt should be made to isolate the diseased birds and carry out thorough disinfection. Cold water may be sprinkled on the head and calomel given in doses of 0.02-0.05 gm.

**Literature.** Alexander & Tandler, A. f. Ohrenheilkunde, LXVI, 161.—Barany, Neur. Cbl., 1906, 776.—Bergmann, Fortschr. d. Vet. Hyg., 1907, 242 (Lit.).—Beyer, A. f. Ohrenheilkunde, LXIV, 273.—Beyer & Lewandowsky, Engelmann's A. f. Phys., 1906, 451.—Dexler, Z. f. Tm., 1897, I, 124.—Klee, Gefügelkrkh., 1905, 32.—Zürn, D. Z. f. Tm., 1875, I, 278.

## 5. Paralysis of the Vagus.

**Occurrence.** Apart from paralysis of the recurrent nerve, paralysis of the vagus is of very rare occurrence owing to the protected position of the nerve.

**Etiology.** Inflammatory processes in the vagus are sometimes set up by bacteria or their toxins, or the nucleus or root of the nerve may become diseased owing to disease of the medulla oblongata. Thomassen has adduced experimental evidence that in the horse chronic lead poisoning causes extensive paralysis of all the branches of the vagus. The various species of *Lathyrus* appear to be capable of producing a similar condition.

**Symptoms.** Since the greater part of the pharynx and esophagus are supplied with motor fibers from the vagus, difficulty in swallowing is a prominent symptom; there may be regurgitation of food or it may collect in the esophagus. Paralysis of the muscles of the larynx causes stertorous and difficult breathing. The animals cannot be made to cough, but when coughing occurs naturally it is deep and prolonged. If attempts be made to make an animal cough there may be simply a prolonged forcible expiration. As a result of the difficulty of swallowing and loss of sensation of the mucous membrane of the larynx, food may pass down the trachea and may cause death by suffocation or more frequently gangrenous pneumonia. The pulse is accelerated and in cases of lead poisoning it is hard. There are also in all probability disturbances of the movements of the stomach and intestines, but nothing definite is known at the present time regarding this.

**Treatment.** This is, as a rule, useless. In cases of chronic lead poisoning the systematic administration of potassium iodide may be tried.

**Literature.** Thomassen, Monatschr. f. Psych. u. Neurologie, 1903, 423 (Lit.).

## 6. Paralysis of the Recurrent Nerve.

### *(Roaring, Laryngeal Hemiplegia.)*

**Historical.** The connection between roaring and paralysis of the recurrent nerve was first demonstrated by Dupuy (1830) and Youatt (1833) showed that the paralysis of the nerve is followed by atrophy of the muscles supplied by it. The true nature of the disease was first explained fully by Günther (1834). Within recent years (1902) the experiments of Thomassen and the histological investigations of Lührs (1904) have added considerably to our exact knowledge of the condition; and the clinical observations of Dieckerhoff (1900), and the work of Schindelka and Polansky (1889), and of Malkmus (1897) have also added greatly to our knowledge of the subject.

**Occurrence.** Roaring is exceedingly common in horses in England, France, Hanover, Holstein, Austria and Hungary and in countries in which horses, and especially thoroughbreds, are bred in conjunction with intensive agriculture. In the Argentine Republic and in South America generally, in Egypt, India, Arabia and Australia the disease is rarely met with (Nocard, Fleming). Isolated cases occur in the other species.

**Etiology.** Recent investigations appear to support the view that in the immense majority of cases infectious diseases are to blame for the causation of recurrent paralysis. Experience shows that the disease is a common sequel to strangles, pneumonia or infectious sore throat, and it is worthy of notice that in all these three diseases the streptococcus equi is more or less concerned. The effect of strangles is shown clearly in that in the Argentine Republic, where strangles and pneumonia are not of common occurrence, paralysis of the recurrent nerve is only exceptionally observed (Nocard), while the part played by pneumonia is shown by the records of the Prussian army in which 0.4 to 2.8 per cent of the animals affected with pneumonia afterwards become roarers. In these cases the paralysis is principally due to inflammation caused by bacterial toxins circulating in the blood. In a case recorded by Fröhner there was paralysis of the nerve due to dourine.

It was shown by the histological investigations of Thomassen that there was a degeneration of the nerve fibers which reached a maximum in the immediate neighborhood of the larynx and that the alterations gradually became less pronounced in the central direction until they had entirely disappeared in the nerve in the lower portion of the neck or in the chest cavity. There were no changes in the fibers running in company with the vagus itself. Apparently the toxin tends to affect the distal portion of the nerve. In many cases of pneumonia the paralysis may be produced by extension of inflammation in the left thoracic cavity to the nerve lying in the subpleural connective tissue.

In a proportion of cases a causal connection can be traced between the disease and certain poisons. Thomassen proved experimentally beyond a doubt that in the horse lead is capable

of causing degeneration of all the branches of the vagus, the degeneration being most pronounced in the terminal portion of the left recurrent nerve. The observations of Beckmann and Schmidt yielded the same results; animals working in the neighborhood of lead works are affected in large numbers. In rare cases the disease may be caused by long-continued feeding on certain foods, riga pea, chick pea, lucerne. Whether the disease in these cases is due to a lesion of the nucleus, as supposed by Leather and Cadéac, or to a peripheral disease, is not definitely known. In view of the fact that the foods mentioned are not generally responsible for any such effect it must be supposed that they are capable of affecting the nerve injuriously under certain circumstances only. Verrier was able to demonstrate the toxic effect of the *Lathyrus cicera* experimentally, while Agonigis' experiments were negative. The cause of the suddenly occurring paralysis observed by Friis and Müller in a number of horses after feeding on molasses is quite unknown.

Other causes play a small part in the production of the disease. Diseases of this kind are observed more frequently in the other species. Aneurism of the aortic arch may cause pressure on the nerve, as may also tumors at the entrance to the chest, in the neighborhood of the aortic arch, or the trachea, and one branch is as likely to be subjected to pressure as the other. Enlarged lymphatic glands must be considered in this connection and also malignant growths, suppurative inflammation, leucemia and pseudo-leucemia. It is only rarely that the cause of the pressure is enlargement of the thyroid, dilatation of the esophagus or abscess in the neck.

The recurrent nerve rarely suffers traumatic injury, and when this does happen it is generally the more superficially placed nerve on the left side; for example in opening an abscess (Kühnert) or venesection (Günther). Huth explains the common occurrence of the disease among geldings on the supposition that the nerve on the left side is injured mechanically while the animal is thrown for the operation of castration. This view, however, is not in agreement with the manner in which the disease develops and its course.

In many cases rheumatism is supposed to be the cause, but even in these cases the coincident effects of infective materials are not excluded.

Finally, bulbar paralysis is generally followed by paralysis of the recurrent nerve. Supra-nuclear paralysis has not as yet been observed in animals.

Experience shows that in about 95 per cent of cases the paralysis is on the left side. Attempts are made by many authors to explain this by the peculiar anatomical disposition of the left recurrent nerve.

The nerve on the left side is considerably longer than that on the right. It passes right into the thorax and turns round the arch of the aorta, while the right



nerve passes round the costo-cervical artery. The nerve on the left side is covered by the pleura almost up to its point of exit from the chest, and in the neck it is placed much more superficially than the right nerve. There appear to be grounds for the supposition that the longer nerve is more exposed to unfavorable influences than the right. According to Martin the conductivity of the nerve may be disturbed by pulsation of the aorta if that is excessive, or if the nerve is in close contact with a dilated arch. According to Ellenberger the fat around the aorta disappears when a horse is in training and consequently the recurrent grasps the aortic arch more closely. Some authors state that a long neck is a predisposing influence. They hold that if the neck be very long the nerve is stretched and pressed more firmly against the aorta. Finally, according to Martin, the backward displacement of the heart during development of the other organs must be compensated for by the nerves.

Thomassen's investigations furnish a more satisfactory explanation of the facts observed. In the great majority of cases it must be supposed that the left recurrent nerve is far more susceptible to toxins and infective materials. Experience shows that in poisoning with riga pea or lead it is principally or exclusively the left nerve that is affected and, further, in the apparently primary cases of roaring there is inflammation only in that portion of the nerve which is outside the cavity of the chest, well away from the influence of the circulatory system. The reason of the greater susceptibility of the left nerve to toxins is not yet known.

It appears to be very doubtful whether there is such a thing as roaring due to a primary disease of the muscle, if one excepts the case (Glöckner) in which there was thrombosis of the carotid. The coincident disease of the muscles suspected by Nocard is quite possibly the result of degeneration of the nerve set up by the virus of strangles. On the other hand the cases of recovery that are considered by some authors to furnish proof of the myopathic origin of the disease in reality do not furnish any such proof since recovery of the nerves may take place.

It is generally admitted that a tendency to the disease is hereditary, and investigations confirm this view. Cadéac and Nocard rightly point out that the frequent occurrence of the disease in certain studs may be due to the frequent occurrence of strangles and pneumonia. Thus, in the Argentine Republic where roaring occurs very rarely, imported stallions that are roarsers are used for breeding without the tendency to the disease being transmitted to their offspring. Certain breeds, especially well-bred animals, and English thoroughbreds appear to be more liable to become affected than half-breds or others. Age appears to exercise some influence on the occurrence of the disease, the majority of cases occurring in animals between the ages of 3 and 6 years. Old horses are affected with moderate frequency, but it is very rare to find an animal affected during the second or third year. Sex appears to have no influence on the occurrence of the disease.

Müller has seen the disease repeatedly in the dog. In one case, the animal had had an attack of broncho-pneumonia, due to distemper five months previously; while another case was caused by dragging on the collar while being led. Frick records a case due to a fall out of a window, and in a case recorded by Albrecht, the cause was not discovered. Paralysis of the recurrent nerve has been observed in cattle by Vitz, Ollmann, Besnoit and Prietsch, the cause being enlargement of the peribronchial lymphatic glands.

**Pathogenesis.** Since the recurrent nerve is a mixed nerve the excitability of the sensory fibers is first increased, the increased irritability being in proportion to the rapidity with which the process develops. If the disease develops slowly, there may be only very slight irritability of these fibers. Sooner

or later the motor fibers lose their conductivity with resulting paralysis and atrophy of the muscles supplied by them. For some unknown reason it is only the nerve fibers supplying the muscles dilating the glottis that are affected in the first instance, but afterwards the others may become paralyzed. In exceptional cases the muscles constricting the glottis are first attacked, or both dilators and constrictors may be affected simultaneously. According to Malkmus this is the common condition. As a result of the infranuclear paralysis and consequent relaxation of the muscles, the vocal cord on the affected side does not move at all or only slightly, but is drawn towards the middle line by the unaffected constrictor of the larynx. It may, however, be forced into the lumen of the larynx together with the arytenoid cartilage by a forcible inspiration, but in either case there is stenosis of the larynx. Paralysis of the muscles causing closure of the glottis destroys the power of coughing. In time the conductivity of the sensory nerves is lost with the result that there is loss of sensation in the mucous membrane of the diseased side, and also to some extent of the opposite side, because some fibers of the nerve may pass from one side to the other.

**Anatomical Changes.** In the early stages and in slight cases the only recognizable lesions are pale yellow streaks in the posterior crico-arytenoid muscle. These streaks may also be present to a less extent in the lateral crico-arytenoid muscle and the transverse arytenoid. In severe cases there is degeneration of a number of the fibers, and finally the muscle may be pale yellow or grayish-red in color and flattened in shape. If the recurrent nerve be exposed the reduction in the size of the nerve and its gray color indicate the degeneration that has occurred, but as a rule, the first exact information as to the diseased condition of the nerve can be obtained only by histological examination.

According to Süßdorf the portion of the nerve lying between the aortic arch and the trachea is normally flattened. Lührs states that the left nerve is thinner than the nerve on the right side.

**Symptoms.** The most striking symptom is stridor in the larynx during inspiration. The sound varies in different cases and with few exceptions is heard only during movement. The character and intensity of the sound depend upon the degree of the stenosis, and continued exercise or great exertion are determining factors in the production of the sounds. Thus in a slight case great muscular exertion, such as galloping on soft ground, causes only a slight whistling sound, while in the later stages of the condition, in severe cases, or in cases in which the constrictors are perfectly or practically healthy, while there is paralysis of the dilators of the glottis a few moments' trotting are sufficient to cause a much more audible

sound. Now and then, cases are met with in which the particular sound is audible even while the animals are at rest, or from the slightest stimulation of any kind. Cases are very rare in which owing to bilateral paralysis there is a sound during expiration due to stenosis of the glottis. It is easy to prove that the sound is influenced by the quantity of air entering the trachea and the rapidity with which it is taken in. If one nostril of a roarer be closed or both of the nostrils are partially closed the noise due to the stenosis disappears immediately. In this lies the explanation of the fact that the sound ceases immediately after the animal stops, or at the latest within 5 to 8 minutes after.

Respiration is more or less difficult during severe exertion. Simultaneously with the appearance of the sound, symptoms of dyspnea set in, the nostrils are opened out in a trumpet-like manner, the ribs are raised spasmodically, the flexible portions of the chest wall sink inwards at each inspiration, and the anus moves in and out with each inspiration and expiration respectively. If the work be continued there may, in severe cases, be symptoms suggestive of suffocation and the horse may fall down. The difficulty of respiration soon disappears, and within one to five minutes after the animal stops, there may be only as much deviation from quiet respiration as is seen in sound horses under the same conditions.

During rest symptoms are either very slight or quite absent. In somewhat more advanced cases it is often difficult to make animals cough, but the cough in such cases is deep, prolonged and rattling. This is proof that the glottis cannot be closed and the paralyzed and relaxed vocal cord makes slow oscillations. So long as the vocal cord is sufficiently tense to allow of closure of the glottis the cough does not show the characters described. In many cases the neigh is more hoarse.

Since the irritability of the sensory nerves tends to be exaggerated before conductivity is lost there is a dry cough which is not associated with any discharge from the nose before the onset of paralysis and for some time after. This cough can be caused far more easily than normally. Some authors have observed catarrh of the larynx before the onset of symptoms of paralysis.

In many cases the arytenoid cartilage of the left side can be pressed more easily into the cavity of the larynx, by palpating the larynx, causing a whistling or rattling inspiration. Palpation of both sides frequently reveals atrophy of the muscle on the left side.

Examination of the larynx with the laryngoscope shows that one vocal cord, generally the left, moves only slightly during respiration, or remains motionless in the middle line, or in cases in which the dilator alone is paralyzed it remains vertical. In cases of bilateral paralysis both the vocal cords move sluggishly or remain motionless. In view of the fact that with the laryngoscope a bird's-eye view of the larynx is obtained, slight dropping of the arytenoid cartilage cannot be deter-



mined, but it may be suspected in many cases, owing to asymmetry of the larynx, the ary-epiglottic fold appearing shorter and curved in its hinder part.

Immobility of the vocal cords caused by paralysis must not be confused with the position occupied by them during prolonged inspiration, for in the former case they are unable to make any movement, while in the latter pressure on the nasal septum, blowing into the nose, drawing one or two straps tight round the thorax are sufficient to set the vocal cords in motion again (Malkmus).

According to Bassi the condition of the vocal cords in horses can be examined by introducing a brilliant light into the posterior portion of the widely opened mouth. This plan although useful in small animals is impracticable in the horse, the different portions of the larynx not being visible owing to the length of the soft palate.

**Course.** In the great majority of cases the disease is chronic. It is only very occasionally that the condition appears suddenly with pronounced symptoms of illness, persists for several weeks and then gradually disappears or becomes chronic. Such cases have been recorded by a number of authors (Günther, Lies, Vollers, Alberts, Albrecht, Malkmus, Friis, Müller and others). Malkmus, from the findings in one case carefully examined with the laryngoscope, describes a complete bilateral paralysis as opposed to the usual unilateral chronic paralysis, in which there is a possibility of recovery. With the exception of these very rare cases the progress of the disease is slow. Symptoms appear at the most four to six weeks after the commencement of the disease process and reach their maximum after a long time, sometimes one to two years. Sometimes paralysis makes its appearance ten to eleven days after the appearance of the primary disease (Rosenfeld, Fröhner). The sound due to stenosis increases from this time onwards, in constantly shorter intervals during work, until a few paces are sufficient to cause a loud roaring and symptoms of dyspnea.

**Diagnosis.** Paralysis of the vocal cords or their muscles can be determined with certainty only by examination with the laryngoscope, and in small animals by direct inspection. If it is possible to press the arytenoid cartilage easily into the larynx or if atrophy of the muscle on the outer surface of the cartilage can be detected one may form the opinion that the disease is in existence, but this can only be done in advanced cases, and the possibility of error is not excluded. Coughing is of only slight value from the point of view of diagnosis because it is difficult or impossible to make many sound horses cough. If paralysis exists already, the cough may not be specially characteristic, and on the other hand, there may be the peculiar type of cough in other diseases of the larynx.

With a probability that amounts almost to certainty the disease may be diagnosed when some other disease is or has been in existence which experience has shown may be followed by paralysis of the recurrent nerve (strangles, pneumonia, neoplasms, aneurism, etc.). In the absence of such information

and without inspection of the interior of the larynx one can diagnose with certainty only some stenosis of the upper air passages or larynx, generally termed roaring, but only a conditional opinion can be expressed as to whether paralysis of the recurrent nerve is the cause of the condition. According to Cadéac and Fleming, whistling and roaring are caused by paralysis of the recurrent nerve in 95 to 99 per cent of cases.

Whistling and roaring during work may be due to stenosis of the upper air passages from other causes. Neoplasms in front of or in the larynx (tumor-like growths of a glanderous nature, Dexler), dilatation of the guttural pouches, curvature of the trachea, proliferations of the laryngeal mucous membrane, may cause exactly similar symptoms, as may also stenosis of the pharynx or posterior portions of the nasal fossæ. While the last named type of stenosis, and that due to curvature of the trachea or larynx can be detected by careful examination of the exterior, this is not so in the case of stenosis of the larynx.

Stenosis of the larynx may occur in acute diseases, and especially in acute edema of the glottis, or owing to acute swelling of the mucous membrane. If the stenosis is only moderate, roaring and dyspnea are observed during work only. In such cases a diagnosis may be based on the history of the case, the course of the disease, and especially the alternation of exacerbations with improvements.

Temporary paralysis of the recurrent can only be differentiated from the chronic progressive form of the disease by observation of the course taken by the process.

The best method of testing a horse as to whether he is a roarer is to gallop it in a circle on a lunging rein with its head held in and turned slightly to the right. If results are not obtained, saddle horses may be galloped or ridden at a fast trot and harness horses may be driven. If possible the ground should be soft in order to cause violent inspirations and to avoid masking the sound by the noise made by the cart or by the horse's feet. The head should be held well in because this puts the muscles of the larynx in an unfavorable position (Günther) and consequently the noise is appreciable earlier. In view of the fact that under such conditions many sound horses breathe noisily in fast paces, particular attention must be paid to the nature of the sound produced. It is only a high-pitched whistle or a deep roar that indicates stenosis. A sound of this type is never produced by a sound horse.

Further investigations are necessary to decide how often individual muscles of the larynx are paralyzed, and whether the muscles other than the dilators are sometimes alone involved. Thomassen's observation that many horses show disturbance of neighing before there is any whistling appears to indicate that the constrictors only may be affected for a time.

**Prognosis.** Laryngeal paralysis due to some infectious disease or intoxication not rarely disappears within some weeks or months, while the apparently spontaneous disease, or paralysis due to compression of the nerve is constantly progressive and is associated with a decreased power of work or loss in value. The question as to the extent to which the disease affects the animal's capabilities solely depends upon the type of work done. Whereas, a saddle horse is rendered use-

less by a moderate severity of the condition, a draught horse or one that is used at slow paces may last for years.

In the early stages of the disease difficulty of respiration appears even with severe work only after a long time, thoroughbreds are capable of running short races without distress. Thomassen's investigations have shown that age is a factor that comes into play, for disturbances are more severe in young animals, presuming the extent of the disease of the nerve to be the same, and also in addition to the paralysis of the muscles, marked distortion of the whole larynx is more likely to happen in young animals.

**Treatment.** In cases that set in acutely anti-rheumatic treatment or treatment designed to combat acute catarrh of the air passages appears to be indicated.

For the chronic form of the disease, Levi advises intracheal injection of strychnine (0.01 to 0.05 gm. in increasing doses two to three times daily), or Lugol's solution (20 to 30 gm.), but others have not had any results from this treatment. The same applies to the internal administration of preparations of iodine and arsenic.

Lindemann claims to have had recoveries in many acute cases obtained by repeated partial closing of the nostrils, causing more powerful contractions of the dilators of the larynx. In six cases, Tagg united the recurrent nerve with the vagus and in three cases the roaring disappeared.

Günther practiced resection of the sunken arytenoid and of the vocal cord, an operation which was afterwards modified by Stockfleth and Möller. Good results with this operation have been recorded repeatedly, but in a proportion of cases (according to Hirsch in 50 per cent) constriction of the larynx reappears owing to contraction. Hirsch describes arytenoidectomy as a very dangerous operation, and one to be considered only when it is a matter of lessening the dyspnea sufficiently to permit a horse to carry out its usual work, but it does not do away with the loud respiration. More recently, Blanchard has practiced cricotomy or cricoidectomy, and has cured seventeen animals out of thirty-six.\*

Draught horses may be rendered serviceable by performing tracheotomy and inserting a tube, and this plan may be applied to advantage even to race horses. Tracheotomy also appears to have a beneficial effect upon the disease (Albrecht). Bridles which partly compress the nostrils are not to be advised for although they tend to prevent the noise they impair the animal's power.

If exact observations provide actual proof of the transmission of the predisposition in special cases, it is advisable to cease breeding from the animal transmitting it.

\*Recently the Williams operation for roaring appears to give very good results and extensive statistics show that about 70% of recoveries follow the operation. Hobday reported 140 cases operated upon with about 75 permanent recoveries.—(EDITORS' NOTE.)



**Literature.** Agonigi, N. Erc., 1900, 305.—Albrecht, W. f. Tk., 1905, 805, 343.—Beckmann, Z. f. Vk., 1891, 253.—Cadéac, Journ. Vét., 1909, 30.—Dexler, Nervenkrkh. d. Pferdes, 1899, 34 (Lit.); *Ergebn. d. Path.*, 1900, VII, 459 (Lit.).—Friis, *Maanedssk.*, 1905, XVII, 265.—Fröhner, *Monh.*, 1908, XIX, 123.—Guttman, Z. f. Vk., 1908, 80.—Harms, B. t. W., 1906, 97.—Hirsch, Über die Arytänektomie usw. Diss. Leipzig, 1908 (Lit.).—Labat, *Rec.*, 1900, 155.—Malkmus, D. t. W., 1897, 19.—Möller, *Das Kehlkopfpeifen d. Pferde*, 1888 (Lit.).—Müller, S. B., 1892, 25; 1893, 21.—Prietsch, *ibid.*, 1905, 77.—Rosenfeld, Z. f. Vk., 1895, 161.—Tayg, *Journ. of comp. Path.*, 1904, 156.—Thomassen, *Monh.*, 1904, XIV, 193, 289; *Monatsschr. f. Psych. u. Neurol.*, 1903, 423 (Lit.).—Verrier, *Rec.*, 1883, 657.—Vossage, D. t. W., 1900, 209.—Walther, S. B., 1902, 167.

**Spasm of the Larynx.** (Spasm of the Glottis.) References to spasm of the larynx in literature are very scant (Günther, Gerlach, Leblang, Ebinger, Dieckerhoff). Hutyra and Marek recognized one case in a foal by examination with the laryngoscope. In one case recorded by Degive it is probable that the stenosis was due to a growth in the pharynx. The disease is very rare in the horse. Bedel has recorded one case of spasm of the glottis in a dog.

The cause is quite unknown, but it does not appear to be improbable that the condition occurs under the same circumstances as paralysis of the larynx. The condition might be due to some nervous disorder which does not lead to loss of conductivity of the motor nerves. At the present time there are no facts to support the idea that the spasms may be due to a neurosis.

The symptoms agree in the main with those of paralysis of the recurrent nerve, only the noise is in many cases quite unconnected with movement, and may even disappear after long and vigorous exercise (Gerlach). During an attack, closure of the rima glottidis can be discovered with a laryngeal mirror. Hutyra & Marek are of the opinion that this occurs only in horses that have had tracheotomy performed (Authors' case).

In the differential diagnosis of the condition neoplasms of the larynx and pharynx and laryngeal paralysis must be taken into consideration.

In the treatment of the condition an effort must be made to lessen the nervous irritability by means of narcotics (morphine and chloral hydrate). If there be great dyspnea, tracheotomy must be performed.

## 7. Paralysis of the Suprascapular Nerve.

**Occurrence.** Up to the present time this disease has been observed almost exclusively in horses, but in these animals the condition is not very rare. A case in a dog has been recorded by Zimmermann, and a few cases in cattle by Kovács, Bru and Székely.

**Etiology.** Paralysis of the suprascapular nerve is generally due to mechanical injury in the neighborhood of the shoulder. It is caused principally by colliding with some object or another horse, the nerve being injured where it turns round onto the outer surface of the shoulder blade, and is placed practically superficially an inch or two above the

shoulder joint. The nerve may be injured by falling, slipping, when an animal is cast, or through fracture of the scapula. The superficial position of the nerve at the point where it turns round the edge of the bone renders it possible for an injury to happen in any position of the limb, and not, as supposed by Möller, only when the leg is not bearing weight. Zimmermann observed the paralysis in a dog associated with paralysis of the anconeus.

**Symptoms.** Paralysis of the suprascapular nerve causes paralysis of the supra- and infra-spinatus muscles, the deltoid and teres minor. The result of this is that the leg moves backwards and outwards when weight is put on it, and especially when the weight first falls on it. During the whole of the period during which the leg supports weight a gap of variable size is left between the shoulder and the chest wall. The horse is unable to abduct the paralyzed limb and consequently is unable to move over towards that side. While the weight is upon both limbs, no abnormality may be visible. In exceptional cases the paralysis is bilateral. Bru records the occurrence of dropped shoulder in a cow associated with flexion of all the joints below the elbow. At the onset the cow moved on three legs.

The paralyzed muscles degenerate and undergo atrophy about the second week, and this atrophy may be so marked that the muscles are reduced to soft, thin structures, the spine of the scapula becoming very prominent. Experience with peripheral paralysis in other parts permits one to conclude that an electrical and a mechanical degeneration reaction should be demonstrable in the more severe cases.

**Diagnosis.** In the diagnosis of this condition, rupture of the muscles involved or of their tendons must be excluded as these conditions cause similar symptoms.

**Prognosis.** Contrary to the opinion expressed by the majority of authors, who believe that a larger percentage are incurable (Friedberger and Fröhner 75 per cent) or are curable only after several months, Schimmel believes that in the majority of cases a cure can be effected. A general rule can scarcely be laid down in this connection because the injury received by the nerve varies from case to case. Recovery appears to be possible in many cases, as is seen in traumatic paralysis of peripheral nerves in general, but this rarely occurs within a few days or weeks, it is generally only after a period running into months or even a year that recovery is complete. In some cases, the motor disturbances disappear with time, but there is always a certain amount of atrophy remaining. No opinion can be formed at the commencement of the disease as to the result, it depends upon the further course taken by the

disease. The more rapidly the atrophy makes its appearance, and the more rapidly it progresses, the later will be the cure, or there may be no recovery. The animal will only be prevented from moving at a fast pace, walking is not much affected.

**Treatment.** Schimmel's treatment appears to be rational and to have yielded very good results. His treatment consists in massaging the affected muscles for the first two weeks, passively moving the leg after the first fortnight, by extending and flexing it, adducting and abducting, and then from the third or fourth week, giving the horse systematic exercise. Electricity may sometimes be used with advantage, but the various stimulants (strychnine, veratrine, etc.) appear to be almost useless. As a general rule paralysis of peripheral nerves tends to recover without treatment, provided the lesion is not sufficiently severe to exclude the possibility of regeneration.

**Literature.** Bru, *Rev. Vét.*, 1908, 741.—Kovács, *A. L.*, 1907, 495.—Maschke, *S. B.*, 1904, 181.—Schimmel, *O. M.*, 1900, 120 (*Lit.*).—Székely, *A. L.*, 1909, 640.—Zimmermann, *Vet.*, 1897, 576.

## 8. Paralysis of the Radial Nerve.

**Occurrence.** Paralysis of the muscles supplied by the radial nerve is seen with comparative frequency in the horse. In other animals it is far more rarely met with, but nevertheless it does occur, and principally in the ox and dog.

**Etiology.** The radial nerve is easily injured by traumatism at the point where it is almost immediately under the skin, and turns round the bone to reach the outer surface. This point is just above the lateral epicondyle. In horses and cattle the nerve may be crushed through falling, heavy draught, kicks, or blows with the shaft, collisions, etc. Slipping backwards or sprawling the leg forwards may cause injury to the radial nerve. (Nietzold, Castagne.)

In many cases the paralysis appears to be partly due to cold. Bräuer records the occurrence of the condition simultaneously in three horses which were exposed to a cold wind while wet, but the symptoms rather appear to suggest that the condition was one of muscular rheumatism. On the other hand, there is sometimes radial paralysis after influenza. Fröhner and Möller have observed the condition in the dog, probably resulting from distemper. Scoffié and Sérès ascribe the paralysis to neuritis resulting from inflammation of the axillary gland. Hébrant records a case in which the radial nerve was compressed by an enlarged axillary gland. Hansen observed a case in an ox following the injection of tuberculin.

The cause is in some cases situated in the spinal cord. A



case in a horse is recorded by Kutzner in which there was simultaneous paralysis of the left radial nerve (associated with paralysis of the anconeus) and of the crural nerves.

Fröhner is of the opinion that the cause of the condition lies in the muscular tissue and that myositis leading to atrophy and paralysis result from excessive strain of the muscles. The occurrence of muscular paralysis in the horse cannot be denied and the credit of having adduced proof of this principally belongs to Fröhner, but the conclusion must not be drawn from observations in which no exhaustive investigations of the nerves were made that this kind of paralysis is of more frequent occurrence than that of nervous origin. The superficial situation of the radial nerve at the point indicated make it extremely probable that, in the horse particularly, pressure on the nerve is of comparatively common occurrence.

**Symptoms.** In view of the fact that radial paralysis is generally due to an injury of the lower portion of the arm, and that in this situation there is only that part of the radial nerve which supplies the muscles below the elbow joint, traumatic radial paralysis usually shows itself in a paralysis of the extensor muscles lying on the forearm. It is only exceptionally that the sensory fibers supplying the outer surface of the forearm or the motor fibers of the extensors of the elbow joint are involved. If the injury is inflicted at a still higher level, about the level of the shoulder joint or in the vertebral canal, the paralysis involves the anconeus group of muscles.

During rest the shoulder and elbow joints are held extended and the rest of the joints flexed (fig. 106). If the limb be forced back by pressure on the carpus, the weight is borne by the limb and the position does not differ from the normal, but if the animal moves the joints are flexed again and the animal cannot straighten the limb. If the animal be made to walk the limb is advanced by means of the extensor muscle of the shoulder until it is vertically under the body or a little further forward, the hoof wall being dragged along the ground. Directly the weight is put upon the limb all the joints are flexed, because the foot is not properly placed owing to loss of power of the extensors. It is very difficult for the animal to get up and sometimes quite impossible. The extensor muscles are in a condition of relaxation both during movement and rest, and in somewhat more severe cases atrophy soon sets in. The elbow joint is somewhat lower than on the sound side (Plósz). The sensibility of the skin and the distribution of heat are not in any way abnormal. In some cases there is loss of sensation or evidence of pain in the parts where the nerve ramifies (Bossi, Hébrant).

If the extensors of the elbow are paralyzed at the same time the motor disturbances are much more striking and these muscles are also relaxed and atrophy soon occurs.

If the paralysis is incomplete the motor disturbances are less obvious. During rest the animal may, for example, be able to extend the limb to some extent so as to be able to bear some part of the weight on it, but during movement the animal keeps

on stumbling with the affected limb or advances it by jerks at each step. If the paralysis be only slight the motor disturbances are most obvious when the animal is made to move over uneven ground.

In a case observed by Möller a horse lay down much of the time owing to unilateral paralysis of the radial nerve and paralysis of the opposite side developed. Friis records a case in which there was radial paralysis on one side and sciatic paralysis on the other, and Flohil and Kutzner have observed simultaneous paralysis of the radial and femoral nerve in a horse.

**Diagnosis.** The condition is easily differentiated from paralysis caused by thrombosis of the axillary artery by the facts that the pulse can be felt in that vessel and that the paraly-



Fig. 106. Paralysis of the radial nerve (Plósz).

sis is present during both rest and movement. Rupture of the extensor tendons of the forearm can be excluded without difficulty.

**Prognosis.** In the majority of cases the animal recovers and sometimes within a few days, but if the paralysis has been in existence for several weeks and there is already considerable atrophy of the muscle recovery is still possible, but paralysis is often persistent in such cases.

**Treatment.** Recovery may be hastened by applying the treatment already mentioned on page 739.

**Literature.** Albrecht, W. f. Tk., 1903, 594 (Lit.).—Bräuer, S. B., 1890, 74.—Castagné, Pr. Vét., 1903, 387.—Cinotti, N. Erc., 1905, 266.—Diem, W. f. Tk., 1906, 503.—Flohil, Tidsskr., 1904, 486.—Fröhner, Monh., 1897, VIII, 499.—Hébrant, Ann., 1905, 417.—v. Kukuljevič, B. t. W., 1905, 714.—Kutzner, Z. f. Vk., 1904, 492.—Lutz, Monh., 1903, XIV, 532.—Nietzold, S. B., 1904, 79.—Williams, Vet. Journ., 1906, 763.—Wyssmann, W. f. Tk., 1904, 645 (Lit.).



**Radial Cramp.** This condition is frequently seen in dogs that have suffered from distemper. The muscles supplied by the radial nerve show more or less rhythmic contractions. In one particular case, the nerve was surrounded by a callus involving both the bones of the forearm at the distal end. In a condition of rest the toes were extended and the slightest movement caused an exaggeration of the extension, and rendered the outlines of the tendons clearly visible. An animal which had ankylosis of both elbow joints fell down every time it tried to move, and held the fore-legs extended downwards from the elbows. There was no muscular atrophy; and the radial nerve was not painful on pressure.

### 9. Paralysis of the Brachial Plexus.

**Occurrence.** Paralysis of all the nerves of the brachial plexus is a very rare condition in the lower animals, owing to the protected position occupied by it. It is seen most frequently in the small animals.

**Etiology.** In the carnivora and the apes injury to the brachial plexus is likely to happen when the animal is jumping downwards, the axilla coming into contact with some rigid obstacle. In a case recorded by Bayer in which the paralysis was preceded by chorealike spasms, the paralysis was probably of spinal origin. The disease is seen exceptionally in the large animals (Fröhner, Lanzilotti-Buonsanti), and, as in the case of the small animals, may be caused by injuries to or bruising of the shoulder, fracture of bones in the neighborhood, inflammatory conditions of the subscapular connective tissue, or by neoplasms.

**Symptoms.** In cases in which the paralysis is complete the affected limb hangs like a lifeless mass from the body and is trailed along during movement. If the paralysis be incomplete the activity of all or of the majority of the muscles is affected to varying degrees. Passive movements are easily carried out. Sensation is lost either in the entire limb or markedly decreased, or it may be lost only in the areas supplied by certain nerves, but it may be quite normal or even exaggerated.

**Prognosis.** Paralysis due to injury in small animals is likely to be recovered from, but in the case of large animals a cure is rendered impossible owing to the fact that the animals frequently lie down or remain lying down for long periods, and also owing to complications.

**Treatment.** Treatment follows the same principles as that laid down for paralysis of the individual nerves of the brachial plexus.

**Literature.** Fröhner, *Monh.*, 1901, XII, 210.



## 10. Paralysis of the Sciatic Nerve.

**Etiology.** The following causes have been recorded: Kicking (Albert), punctured wounds, falling from a height (Möller), falling (Polfiorow), and myelitis due to distemper. Paralysis of the sciatic nerve associated with paralysis of the fore limb of the opposite side has been observed by Friis in the horse and by Möller in the dog. In a case recorded by Labat the paralysis was due to rupture of a pelvic abscess, and in one by Cadéac to pressure on the nerve by a sarcoma. The disease is observed frequently in dourine owing to neuritis.

**Symptoms.** In complete unilateral paralysis of the sciatic nerve there is loss of power in the lower portions of the biceps femoris and the semitendinosus, and of all the muscles below the stifle joint. The animal is unable to flex the stifle joint, nor can it flex or extend the hock or joints of the foot. In the condition of rest the leg hangs relaxed, but if placed in the proper position is able to support the body because the stifle joint is fixed by the extensors attached to the patella and consequently the lower joints are fixed. There is always marked flexion of the hock. During movement the foot is dragged along the ground and the weight of the body is borne by the toes in the position of plantar flexion. In consequence of this the skin covering the toes becomes abraded. There may be loss of sensation in the portion of the limb below the stifle, but on the other hand there may be hyperesthesia.

If the paralysis be bilateral the picture presented resembles that seen in lumbar paralysis, but differential diagnosis may be based upon the absence of functional disturbances of the bladder and rectum, the normal sensibility of the inner surface of the thigh, the active movements executed by the tail and the hip joint. Atrophy tends to appear early in the paralyzed muscles, as in all cases of peripheral paralysis.

**Treatment.** A cure may at most be looked for if the paralysis be due to an injury. The treatment indicated is that advised by Schimmel for supra-scapular paralysis (see page 739).

**Literature.** Cadéac, *J. Vét.*, 1907, 396; *Pr. Mil. Vb.*, 1901, 104.

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**Sciatica.** In the neighborhood of Saglio in Italy, Giovanoli has frequently observed in goats and cattle, a condition resembling sciatica, due to an inflammation of the sciatic nerve. The condition was thought to be due to cold and badly constructed stables. The stalls which were paved with stone were too short and were provided behind with open drains that were too deep, with the result that when the animals were lying down, the hip joints were subjected to great pressure against the sharp edge of the stalls. In spite of the fact that similar stable construction was found in other places,

no similar symptoms were observed. Serini records the occurrence of a condition resembling sciatica in the dog.

Cattle that are so affected hold all the joints of the diseased limb in a position of moderate flexion, and from time to time draw it slowly up to the abdomen as if trying to ease the pain. They look round at the belly at times, and start at every sound. An attempt is made to prevent passive movements, and if one succeeds in extending the limb passively the animal is likely to fall. Turning round is avoided as much as possible. During movement, the animals hop on the sound limb, the diseased leg being dragged along cautiously. There is pain on pressure along the course of the sciatic nerve. (Giovanoli, Schw. A., 1891, XXXIII, 181.—Serini, Ö. M., 1893, 316.)



Fig. 107. Paralysis of the tibial nerve (Möller).

#### Paralysis of the Tibial Nerve.

Paralysis of the tibial nerve alone is a very rare condition. Only a small number have been recorded, a few in the horse (Möller, Pr. Mil. Vb., 1893) and one in the cow (Schultz).

The animal was unable to extend the hock, and flex the joints of the foot. When the hock is motionless, the affected foot is somewhat flexed (fig. 107), but during movement there is excessive flexion of all the joints; and the foot is put down awkwardly, the movements recalling those of a fowl walking. It is not impossible for the foot to bear weight because the hock joint is fixed by the gastrocnemius tendon. The muscles on the posterior surface of the lower thigh, and the flexor pedis are relaxed and soon atrophy.

#### Cramp of the Muscles of the Tibia.

This condition has been frequently observed in the horse (Strauss, Duschaneck and Hauptmann). The affected leg is extended and directed somewhat backwards, and quite immobile. Neither active nor passive movements can be made. The tendo Achillis is tense and hard; and there is a deep groove between it

and the gemelli. At best, the animal can take only a few steps with the other legs. The cramp is accentuated by mechanical and thermal stimuli. The condition lasts at most from a few hours to a day, but very exceptionally for longer periods. The condition can easily be confounded with outward luxation of the patella. (Duschaneck, T. Z., 1906, 283.—Hauptmann, *ibid.*, 1906, 371.)

**Paralysis of the External Popliteal Nerve.** Paralysis of the peroneus is of very rare occurrence. Fillecke saw a case in a cow that had caught its foot between two boards. Szidon records a case in a dog in which the nerve was subjected to pressure by a shot, while in one case in a dog, the paralysis was caused by creeping under the bed. The condition has been observed in the horse by Darrou, the cause



being pressure on the nerve by a larval *Hypoderma*, while in one particular case the paralysis was caused by pressure on the nerve by a loop in a hobble-shank. Uhart believes that in the case in a horse recorded by him the cause was a false step.

In cases of complete paralysis of the external popliteal nerve the animal is unable to flex the hock and to extend the other joints of the limb. When the leg is advanced the toes drag along the ground; and when weight is put upon the leg, the foot rests with the dorsal surface of the toes on the ground (fig. 108). If the phalangeal joints are extended passively, the leg is capable of supporting the weight properly. According to Günther, the position occupied by the pastern in the horse is more vertical. If the paralysis be incomplete, the animal stumbles a lot during movement, and especially on uneven ground; and all the joints of the affected limb are flexed at the instant that weight falls on the leg. In the later stages, in order to avoid stumbling, the foot is intentionally lifted higher and set down quickly.

There may be no sensory disturbance but in many cases there is anesthesia of the antero-lateral surface of the lower thigh and metatarsus. Atrophy of the muscles and absence of reflexes may be observed.

Paralysis due to injury generally disappears within a few weeks but may persist longer.

**Literature.** Darrou, *Rev. Vét.*, 1902, 586.—Meoni, *J. Vét.*, 1905, 166.—Szikon, *Vet.*, 1898, 129.—Uhart *Rec.*, 1905, 291.



Fig. 108. Paralysis of the peroneal nerve in a case of dourine.

## 11. Paralysis of the Femoral Nerve.

**Occurrence.** Paralysis of the femoral nerve in the lower animals is of very rare occurrence owing to the protected position occupied by the nerve.

Occasionally there is paralysis of the quadriceps femoris in the horse after an attack of hemoglobinuria, which is due to a primary diffuse degeneration of the muscles. According to Fröhner the majority of cases in the horse described as paralysis of the femoral nerve are in reality cases of primary muscular degeneration.



**Etiology.** Injury to the femoral nerve is generally due to over-extension which may be due to slipping or falling with the hind legs spread apart. In exceptional cases the disease may be due directly to a splinter of bone being broken off, or to extravasated blood. Violent kicking may also cause stretching of the nerve. Paralysis following parturient paresis may be due in exceptional cases to stretching of the femoral nerve. Neuritis is sometimes the cause of the paralysis observed in cases of dourine. Other causes are abscesses, hemorrhages or tumors in the psoas muscles.



Fig. 109. Paralysis of the crural nerve with marked atrophy of the quadriceps femoris.

**Symptoms.** Owing to paralysis of the quadriceps femoris the animal is unable to fix the stifle joint when weight is put upon the affected leg and the leg gives way in the other joints at every such attempt (fig. 109). At the same time the stifle joint is not extended sufficiently and consequently the leg is not advanced as far as normally. The patellar reflex is either absent or exaggerated. There is a tendency to loss of sensation of the skin on the inner surface of the thigh, and if the paralysis persists there is atrophy of the quadriceps muscles.

**Prognosis.** If the paralysis is due solely to stretching of the nerve and there is no atrophy of the muscles, recovery may

be expected within a few weeks. In other cases animals learn after some months to walk with safety, the atrophy of the muscles meanwhile gradually disappearing totally or in part. Paralysis due to pressure on the nerve tends to persist for long periods.

**Treatment.** Massage should be practiced and systematic exercise should afterwards be given (see page 739).

**Literature.** Fröhner, Monh., 1897, VIII, 499.—Flohil, Tidskr., 1904, 486.—Grunth, B. t. W., 1904, 93.—Kutzner, Z. f. Vk., 1904, 492.—Schimmel, Ö. M., 1901, 469.

## 12. Paralysis of the Obturator Nerve.

**Etiology.** The cause was not known in two out of three cases in horses recorded by Schimmel, while the disease resulted in a dog that jumped down from a height and fell over backwards. Thomassen records one case, and Willis two, in horses in which the paralysis was due to a callus of the pubis, and in a case recorded by Nocard the nerve was crushed in a fracture of the pelvic bones. The disease tends to appear in dourine.

**Symptoms.** During rest the position occupied by the limb may be normal or there is more or less abduction. This abduction is more marked at a walk and still more at a trot, the animal hopping on three legs, and holding the affected leg in the air in a position of abduction. In some cases there is marked flexion of the hip joint and the stifle moves upwards and outwards. The step is shortened and consequently the animal moves obliquely towards the opposite side. It is very difficult for the animal to back, the affected leg is moved backwards with difficulty only and is abducted during the movement. Adduction of the leg is impossible.

The absence of other motor disturbances indicates simple paralysis of the obturator nerve and subsequent atrophy lends support to the diagnosis. Up to the present no sensory disturbances have been observed.

**Treatment.** Recovery may be hastened by massage of the paralyzed leg, followed by passive movements and systematic exercise from about the third or fourth week (see page 739). Recovery almost always occurs unless the nerve be subjected to pressure.

**Literature.** Rexilius, Z. f. Vk., 1905, 72.—Schimmel, Ö. M., 1894, 387; 1902, 242.—Thomassen, Monh., 1901, XII, 367.

### 13. Paralysis of the Sacral Plexus.

One case each of this kind has been recorded by Thomassen and by Cadéac in horses in which there was gradual atrophy of the muscles of the near hind leg, and also very marked atrophy of the muscles of the left side of the croup. In Thomassen's case the diseased leg was lifted quickly upwards and inwards, and put down clumsily, there being flexion of the fetlock joint. The left half of the croup, the outer and posterior surfaces of the thigh, and the portion of the leg below the stifle were insensitive. Electrical and mechanical stimulation of the muscles were without effect. In Cadéac's case the animal lay for the most part on the sound side, or was unable to rise after lying on the diseased side. Thomassen found marked thickening of all the nerves of the sacral plexus, an increase in the amount of epi- and perineural connective tissue, with atrophy of the nerve fibers. These lesions were probably caused by over-stretching of the plexus. On the other hand Cadéac showed that the plexus was enclosed in a sarcomatous growth. Günther states that he has seen several cases of paralysis of the sacral plexus in the horse.

*Literature.* Cadéac, *J. Vét.*, 1907, 396.—Thomassen, *Monh.*, 1901, XII, 145.

**Paralysis of the Gluteal Nerve.** Subsequent to a difficult parturition, Cuny (*J. Vét.*, 1907, 652) observed rapidly progressing atrophy in the area supplied by the superior gluteal nerve in a mare. The author believed that it was due to crushing of the nerve during the parturition. There was no evidence of motor disturbance.

**Paralysis After Parturition.** This condition is most often observed in cows from five to eight years old. According to Hess, it occurs more frequently in spring and summer and is intimately connected with the parturition. According to this author, it occurs usually after easy and rapid parturitions, while other authors associate it with difficult parturition, supposing that the paralysis is due to compression of the sacral plexus, the sciatic, and obturator nerves, caused by luxation of the sacro-iliac articulation. On the other hand, Franck and more recently Hess, state that the paraparesis is produced reflexly by an injury of the genital passage. This view is supported by the fact that a similar condition can be produced in the rabbit by compression of the uterus. Possibly the paralysis may be caused by luxation or contusion of the hip-joint and also by exhaustion (Hutyra & Marek). The cases occurring after easy parturition, and uncomplicated cases, are scarcely distinguishable from parturient paralysis; or they may be the same thing (Hess, Albrecht, Zehl).

The first symptoms make their appearance either directly after parturition or towards the end of the first day. In exceptional cases their appearance may be delayed beyond this. There is weakness of the hind quarters, and the animals lie down. Once down, they are unable to rise again. Otherwise the animals appear normal.

**Treatment.** If the animal does not get up of its own accord by the fourth or fifth day, it may be made to rise by rubbing its tail between two round pieces of wood, pouring water in its ear, hoisting it up with



hay-bands, by pushing hay or faggots under it. If convenient, slings may be used. Hess, Albrecht, Zehl and others have found inflation of the udder with air, useful.

**Literature.** Albrecht, W. f. Tk., 1906, 741 (Lit.).—Hess, Schw. A., 1905, XLVII, 279.—Zehl, B. t. W., 1908, 117.

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**Paralysis Before Calving.** This is a symptom of weakness, and is seen in old cows that are in poor condition, or in cows that are weak through long retention in stalls. It may be the result of fractures, luxations, bruising, brittleness of the bones, or infiltration of the muscles of the croup or pelvis.

#### 14. Paralysis of the Pudic Nerve.

**Occurrence.** Paralysis of the pudic nerve appears to be a very rare condition and it occurs exclusively in the horse.

**Etiology.** Paralysis of the penis may be due to traumatism (a kick in the perineum, bruising against the bale, etc.), more frequently it is associated with influenza (Barrier, Fröhner, Schulze). In some cases the cause cannot be discovered. In one case Barrier observed, in addition to bruising of the perineum, signs of chronic interstitial neuritis in the pudic nerve. Fröhner is inclined to attribute the paralysis following influenza to a disease of the spinal cord. In a proportion of cases the cause of the paralysis appears to be in the muscles.

**Symptoms.** Owing to paralysis of the retractor penis which is supplied by the pudic nerve there is protrusion of the penis which later becomes edematous because of the obstacles offered to the flow of blood and lymph.

In some cases recovery takes place within a few weeks or months. In a case recorded by Röder recovery took place without any treatment after five years. In the majority of cases the paralysis does not appear to regress.

**Diagnosis.** Protrusion of the penis owing to surgical conditions must be excluded before the disease is taken to be of nervous origin.

**Treatment.** If the paralysis does not disappear after several months amputation of the penis is indicated. This is often followed by stricture of the urethra.

**Literature.** Barrier, Rec., 1899, 70.—Boos and Römer, D. t. W., 1899, 348.—Fröhner, Monb., 1898, IX, 1; 1904, XV, 217.—Hoeg, Maanedsskr., 1899, XI, 209.

### 15. Combined Paralysis of the Tail and of the Sphincter.

In the horse a condition is sometimes observed which is characterized by paralysis of the sphincters of the anus and bladder with simultaneous paralysis of the tail. The cause of this condition is a chronic interstitial neuritis of the cauda equina.

**Occurrence.** The disease, which has been known for a long time, is confined to the horse. Marek's observations do not confirm those of several authors who state that the disease occurs most frequently in mares. In Marek's observations 50% of the cases were in mares.

**Etiology.** Chronic inflammation of the cauda equina is caused by mechanical influences. This may be severe and act only once, or it may be slight and be in action for long periods or come into action repeatedly, and may involve the mobile caudal vertebræ or even the sacrum.

Wolff has frequently seen the condition result from fracture of the caudal vertebræ and Marek has seen one similar case. In a case reported by Rubay fracture of the sacrum led to chronic inflammation of the cauda equina, and in one observed by Kuske and Marek the horse received an injury to the croup during transport by rail. Marek observed the occurrence of the disease in a mare shortly after copulation, in two horses after falling on the buttocks, and in a further case a horse that was in somewhat poor condition had to be helped to rise by lifting on the tail. In a case observed by Mayerstrasse there was also facial paralysis but this in all probability was due to bruising while the animal was down.

Injuries to the croup or tail may obviously lead to bruising and consequent temporary paralysis of the caudal nerves or may lead to the production of clinical symptoms of neuritis of the cauda equina.

**Anatomical Changes.** The nature of the disease was elucidated by the thorough investigations of Dexler and his results have been confirmed completely by other authors (Cadéac, Raymond, Rubay, Hutyrá and Marek). According to these investigations the nerve roots in the vertebral canal posterior to the end of the spinal cord and outside the dura mater are embedded in a fibrous connective tissue which takes the form of an elongated and often asymmetrical swelling, filling up the sacral canal (figs. 110-111) and which extends into the intervertebral spaces. In many cases one or more nerve trunks leading to the brain are wholly or partly free but appear much thickened. As a rule the inflammation does not extend further forwards than the second or first sacral nerve roots, but in a few cases the last two lumbar nerve roots were involved. Exceptionally the process involves the subdural section of the nerve roots and even the pia mater of the cord. In recent cases the connective tissue appears gelatinous, red in color or beset with numerous hemorrhages (fig. 110).

Under the microscope it is seen that the nerve fibers are closely surrounded by newly formed connective tissue. This tissue is very finely fibroblated and at

places, especially around the vessels, the walls of which are much thickened, it is infiltrated with cells. In most of the nerve fibers the medullary sheath is degenerated and in many bundles no fibers can be made out. The cells of the spinal ganglia show various stages of chromatolysis and destruction of the nuclei. The spinal cord appears normal and in a single case only could ascending degeneration of the posterior column be made out with certainty where the process involved the intradural portion of the nerve roots and the last two lumbar nerves were also diseased.

The muscles of the tail appear for the most part degenerated and in more advanced cases somewhat similar lesions have been found in the muscles of the croup. Of the peripheral nerves the following are usually in a condition of degeneration; the nerves of the tail, the pudic nerves, the superior and inferior gluteal nerves, the posterior cutaneous branches of the dorsal branches of the sacral plexus and the posterior hemorrhoidal nerve.

**Symptoms.** In the early stages the sensory nerves are stimulated by the connective tissue which gradually increases in amount and contracts around them, with the result that the skin of the tail and the perineum are hyperesthetic. In consequence of this the animal rubs these parts against any rigid object and, finally, there are symptoms of great restlessness. In a case recorded by Dexler the onset of the disease was marked by severe and persistent priapism. The attendants do not as a rule notice this and the rubbing of the tail is put down to other causes. The matted condition of the hair on the root of the tail indicates that the animal has suffered from severe irritation. In one case (Hutyra and Marek) spasmodic contractions of the gluteal muscles were observed if a loud noise were made or a person approached the animal.

The other symptoms are far more noticeable. The most prominent is paralysis of the



Fig. 110. Terminal portion of the spinal cord with the cauda equina from a case of paralysis of the tail and sphincters. L5-L6 last two lumbar nerves. S1-S5 sacral nerves, of which numbers 4 and 5 are enclosed, together with the first two coccygeal nerves. C1-C2 and the filum terminale, in an elongated, thickening which is much larger on the right side. Between S5 and C2 there was marked congestion and some hemorrhages were also present. The specimen was obtained from the horse shown in fig. 112.



tail. At the trot this hangs lifeless between the buttocks and swings from side to side. At rest it is quite motionless or, if the paralysis remains unilateral for a time it may be moved to one side only. During defecation and urination it is not lifted. As a result of atrophy of the muscles, the tail appears thinner than normal and in the later stages there is a depression between the posterior and upper gluteal regions. As a rule the atrophy is not symmetrical on both sides of the body. The tail offers little or no resistance to passive movements to one or both sides. The paralyzed muscles show electrical degeneration reaction or the absence of electrical or mechanical irritability (Dexler).

There is usually loss of sensation of the skin, the joints and muscle of the tail, the perineum, root of the tail, posterior gluteal region, mucous membranes of the rectum and vagina on both sides, though perhaps not to the same extent. One side

alone may remain insensitive for a time (figs. 112-133). Pricking the muscles or twisting the tail causes no reaction. Similarly, heat, cold and electrical stimulation are without effect. In the later stages there is usually a narrow hyperesthetic zone bounding anteriorly the anesthetic area (Hutyra and Marek), or there may be circumscribed hyperesthetic areas. This hyperesthetic zone gradually advances with the extension of the area of anesthesia in the forward direction. The anesthetic area merges into the area that is hyperesthetic or of normal sensibility in many cases through a very narrow zone that is hypoesthetic.



Fig. 111. Combined paralysis of the tail and sphincters. Cross section through the cauda equina shown in fig. 110 after fixation in Müller's solution. The dark areas mark the position of the nerve fibers which are surrounded by a large amount of connective tissue.

There is marked retention of the feces and defecation may be impossible. As a rule balls of feces are passed only during exercise. The anal region is soiled with feces and in the case of mares with urine, the sphincter is relaxed, the posterior portion of the rectum is widely dilated and packed with feces. Masses of feces project from the anus and after these are removed fresh masses are pushed on from the anterior portion of the rectum and again fill up the dilated portion (figs. 112 and 113).

For a time micturition is quite normal, but in the later stages there are abnormalities in the manner in which the urine is passed. Both during rest and movement it may be passed in small jets without effort or it may be passed continuously in

drops. Any increase of intra-abdominal pressure or movement causes larger amounts to escape. Pressure on the bladder causes a larger quantity to be ejected in a stream and when the pressure is removed the stream ceases. In this case both the sphincter and the detrusor urinæ are paralyzed. In many cases there is difficulty of micturition due to paralysis of the detrusor urinæ. The disturbances of micturition may in time cause cystitis or may lead to the formation of calculi, which may prove fatal.

There are no sensory disturbances and the appetite remains normal. It is only when large quantities of feces collect

Fig. 112.

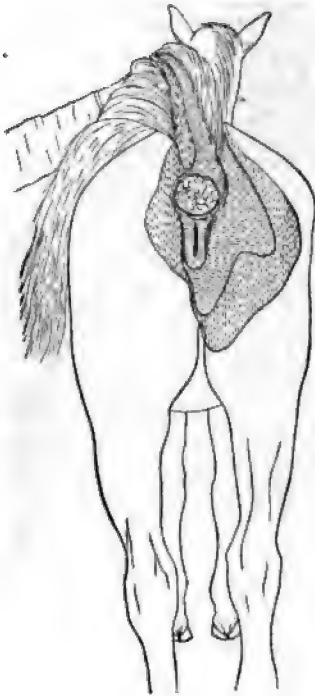


Fig. 112.

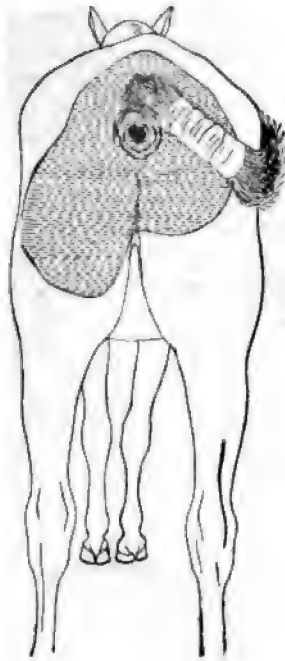


Fig. 112. Combined paralysis of the tail and sphincters. The area marked with horizontal lines was in a condition of anesthesia, while that marked with vertical lines was markedly hyperesthetic. The anus was open and packed with feces. The specimens shown in figs. 110-111 were obtained from this horse.

Fig. 113. Combined paralysis of the tail and sphincters. There was complete anesthesia in the shaded area. The anus was relaxed.

in the rectum that the animals may show slight symptoms of colic and these disappear when the feces are removed. If the masses of feces are not removed the obstruction may be a source of danger.

As a rule there are no motor disturbances, but in exceptional cases in which the process involves the anterior sacral and the posterior lumbar nerves there are symptoms of lumbar paralysis (Hutyra & Marek).

A case is recorded by Holterbach in which a calf showed severe periodical restlessness and gnawing at the root of the tail. In this case the nerves of the tail were compressed by a fibroma.

**Course.** The course of the disease is chronic. The first symptoms generally escape observation, but within two or three months they become pronounced and in many cases remissions are observed. In some cases the symptoms may be fully developed within a month (Marek). With careful treatment the animal may remain useful for a year or more, but it frequently happens that the animal dies before that from cystitis, or there may be lumbar paralysis.

**Diagnosis.** In well developed cases the disease is easily recognizable owing to the characteristic nature of the symptoms. The slow development is sufficient to distinguish it from contusion of the cauda equina or of the posterior part of the sacral portion of the cord (fracture of the sacrum), and the presence of the zone of hyperesthesia distinguishes it from other diseases of the spinal cord. The condition is differentiated from that caused by compression of the cauda equina (Petit) by the discovery of a neoplasm (melanoma) in the sacral portion of the spinal canal.

**Treatment.** The diseased process within the vertebral canal is incurable and consequently the symptoms due to it cannot be removed. With careful treatment, however, an animal may be able to work for a long time. The masses of feces which collect in the rectum must be removed by hand at least twice a day, and the anal region must be kept clean. Suitable treatment must be applied to the catarrhal condition of the bladder.

**Literature.** Dexler, Z. f. Tm., 1897, I, 273 (Lit.); Nervenkrkh. d. Pferdes, 1899, 42 (Lit.).—Holterbach, B. t. W., 1904, 788.—Kuske, Z. f. Vk., 1904, 389.—Marchand & Alix, Rec., 1906, 353.—Marek, Z. f. Tm., 1909, XIII, 33.—Mayerstrasse, B. t. W., 1898, 85.—Petit, Bull., 1906, 266.

## 16. Polyneuritis.

Under this term are included inflammatory conditions of the nerves which involve several nerve trunks, either simultaneously or one after the other, but which are due to the same internal cause.

**Etiology.** Investigation of a number of cases of dourine (Marek) showed that the symptoms were due to an interstitial inflammation of the nerve trunks which extended as far as the dura mater of the cord (see Vol. I). The investigations of Thomassen showed that in chronic lead poisoning in the horse there is parenchymatous inflammation of various nerves. The



nerve that is first affected is the vagus and its branches. The sympathetic suffers to a less degree, and in the nerves of the extremities the lesions are very slight and cause no functional disturbance. Vachetta observed polyneuritis in a fowl as a result of lead poisoning, but Marek was unable to produce the disease by introducing large quantities of lead into the crop. In dourine it is principally the proximal portions of the nerves that are affected, but in lead poisoning it is the distal segments.

An error will scarcely be made if it be supposed that there are other causes of polyneuritis in the domesticated animals. The nervous symptoms seen in chronic mercury poisoning are probably due in part to a polyneuritis, but the question

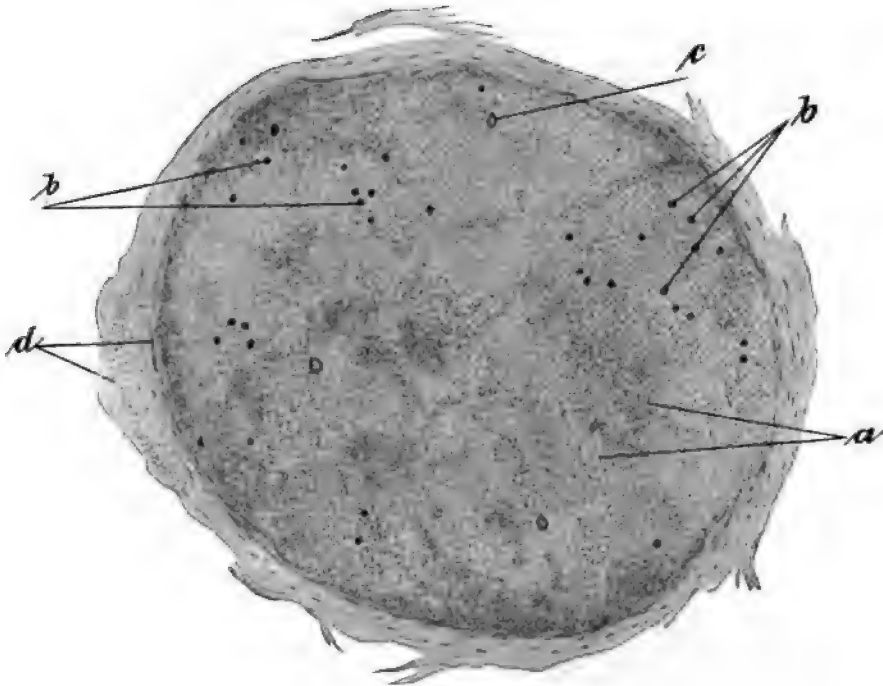


Fig. 114. Cross section of the femoral nerve of a fowl affected with polyneuritis. (a) Extensive infiltration of the endoneurium with mono-nuclear cells. (b) Scattered persisting nerve fibers. (c) Cross section of blood vessel. (d) Slight cellular infiltration of the perineurium abutting on the epineurial tissue.

whether mercury and other metals and viruses can cause polyneuritis cannot at the present moment be answered with certainty. In a case recorded by Lellmann as "infectious polyneuritis" the symptoms were in all probability due to chronic ossifying pachymeningitis of the cord.

In Batavia, Eykmann observed a disease resembling beri-beri in fowls after feeding with cooked rice. The disease had an incubation period of three to four weeks or more, and on the grounds of histological examination he stated that it was a polyneuritis. The disease could be produced experimentally in fowls by prolonged feeding with de-corticated rice; birds of prey and apes were refractory.

The raw unhusked rice was without effect, but decorticated rice warmed to 125° set up the disease just as is the case with rye.

From this fact Eykmann concludes that the husk of the rice and rye contains some protective material which paralyzes the toxic power of the decorticated grains in some way or other. Maurer and Treutlein think that this kind of polyneuritis is due entirely to a chronic poisoning with oxalic acid, large quantities of this acid being produced during the fermentation of the rice in the crop, and this cannot be neutralized by the husk, owing to the absence of calcium salts. In order to produce the disease, the feeding must be continued for several weeks. For this reason the condition described by Kellermann, which was produced by a single meal of rice was certainly not polyneuritis.

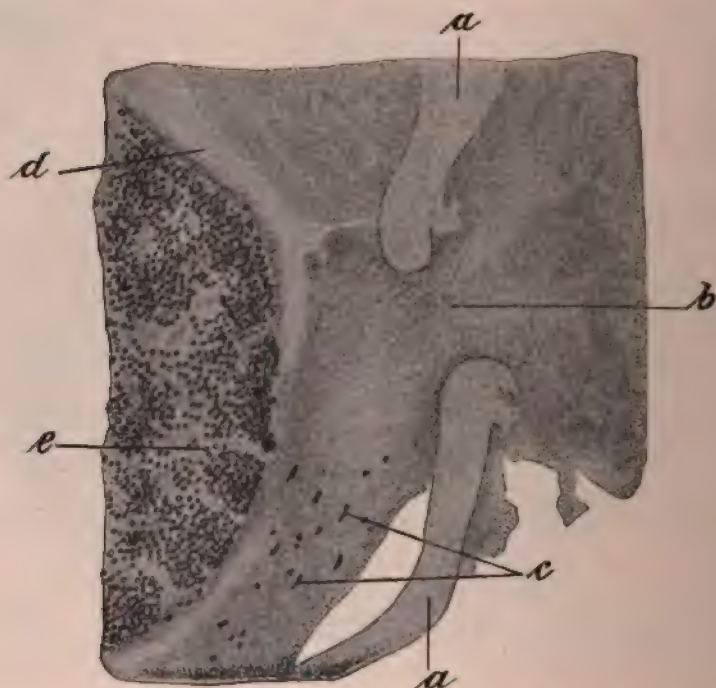


Fig. 115. Cross section of the spinal cord of a fowl affected with polyneuritis. (a) Dura mater showing a nerve root (b) passing through it, which with the surrounding connective tissue appears to be markedly infiltrated with cells. (c) Persisting nerve fibers, some cut transversely and some obliquely. (d) Pia mater infiltrated with cells and from which groups of cells penetrate the peripheral layers of the cord. (e) Focal proliferation of groups of cells.

Eykmann is inclined to think that the disease produced in fowls by Balardini by feeding them on mouldy maize, the symptoms of which were wasting and paresis, was of a similar nature.

Marek observed polyneuritis in cocks, the cause of which was not known, but which was not connected with any particular article of diet. In contradistinction to what was found by Eykmann, polyneuritis was pronounced (fig. 114 and 115), the principal lesion being a chronic interstitial neuritis.

**Symptoms.** The disease occurs in a very pronounced form in dourine (see Vol. I). In cases caused by chronic lead poisoning there is generally only paralysis of the pharynx, esophagus and larynx, acceleration of the pulse, constipation, and sometimes symptoms of colic (Thomassen). It is not, however, quite certain that in many cases there are not symptoms of motor disturbance. In Vachetta's case already mentioned there was obvious extensive paralysis, the fowl was scarcely able to stand and simply squatted. The polyneuritis seen in cases of dourine not rarely disappears without any sequel, but the disease occurring in cases of chronic lead poisoning generally terminates fatally owing to pneumonia caused by foreign bodies.

In polyneuritis of the fowl due to feeding with rice, the gait becomes unsteady, especially when the bird is on a perch, "for it is obvious that if the bird cannot grip sufficiently tightly with the toes, it must exert itself to keep from falling." The birds are not able to get up on their perches, but stand with the legs wide apart, the joints flexed. During movement, the limbs often give way, causing the bird to fall. In the late stages, the birds cannot even stand. The muscles of the wings, body, and neck are paralyzed; there is great emaciation, respiration becomes difficult, and the birds die in five to ten days. In peracute cases death may take place in two to three days.

In polyneuritis of the cock, there is a gradually increasing weakness of the feet; movement becomes more and more difficult, the legs give way under the weight of the body and the birds collapse. As a rule, the legs give way in such a manner that the whole length of the posterior surface of the metatarsus is in contact with the ground. Power of copulating is lost from the outset. In the later stages, there may be complete paralysis of the wings, body, and neck. The course of the disease extends over several weeks. There appears to be no complete recovery. In one cock the paresis lasted unchanged for two years, until death took place from some other cause.

**Treatment.** The treatment of dourine has been dealt with elsewhere (see Vol. I). In cases of chronic lead poisoning potassium iodide should be administered internally, the pasture or food should be changed or the animal should be moved elsewhere in case the food may be the vehicle of the poison. In cases of polyneuritis caused by rice improvement follows a change of food provided it be made early enough.

**Literature.** Kellermann, Á. L., 1907, 631.—Marek, D. t. W., 1901, 417 (Lit. on polyneuritis of chickens).—Thomassen, *Monatsschr. f. Psych. u. Neurol.*, 1903, 423.—Vachetta, N. *Erc.*, 1907, 257.

## 17. Trotting Disease of Sheep.

(*Traberkrankheit* [German], *Tremblante* [French].)

**Occurrence.** This disease, which was practically unknown previously, has occurred with much greater frequency since the end of the eighteenth century. This coincides with the intro-



duction of the Merino breed and with the plan of improving the breeds of sheep to an excessive degree with the object of improving the wool. The disease had been observed previously, for it is mentioned by Leopold in 1750 when great losses were experienced in Spanish sheep, the breeding of which was then greatly increasing and inbreeding was resorted to with the object of improving the breed. Since sheep breeding has been conducted upon more rational lines there has been a great decrease in the number of cases. It appears to be restricted to certain districts and occurs even in these very rarely. At the present time it occurs principally in the pure herds and more rarely among Negretti sheep.

**Etiology.** The actual cause of the disease is unknown.

The disease has been described as exceedingly contagious, not only by breeders but also by veterinarians (Spinola, Haubner, Gerlach, Röhl, and others). There is much evidence indicating that the disease is not hereditary. The principal point is that the disease occurs among sheep that are not pure bred, such as English and German sheep bred for mutton. It is alleged that goats are also attacked. The disease also occurs among pure bred sheep derived from perfectly healthy parents. Fürstenberg records an outbreak that was so severe that almost every animal in a herd of 500 became ill, and one third of them died. Cases of this sort absolutely exclude the possibility of heredity having anything to do with the transmission of the disease. Evidence pointing in the same direction is that the progeny of certain rams become ill in one district and not in another, and further the complete disappearance of the disease from herds that are severely affected when the herds are transferred to other districts. Even this factor was not considered completely satisfactory by the supporters of the theory, other causes being also blamed, such as excessive use of young rams, feeding with rich food or on the other hand, with poor food, sudden changes of food. These factors have not been shown to play any part in the production of the disease by more recent investigations.

The fact that the disease occurs in certain districts, and in some cases in particular parts of these districts, and especially in damp, marshy places (Haubner), suggests that it is due to an infection of some sort. The infection theory put forward by Richthofen has more recently found supporters in Besnoit and Morel, although these admit the possibility that it may be due to an intoxication set up by some food stuff. Cassirer has been unable to transmit the disease to sound animals by the transfusion of blood, but in the blood and cerebro-spinal fluid he found large cocci which, after intravenous inoculation, persisted in the blood of healthy sheep for long periods and then disappeared. One experimental sheep died after a year from exhaustion, without having shown any symptoms of the disease.

A special predisposition on the part of certain breeds of sheep, accords well with the theory of infection. The special susceptibility of the Electoral sheep may be due to the excessive improvement of the breed, to inbreeding and to pampering. Susceptibility to other contagious diseases is seen under similar circumstances. The occurrence of the disease after the introduction of fresh stud animals, and the subsequent spread, are consonant with this theory. On the other hand, a spontaneous occurrence of the disease does not utterly do away with the possibility that the disease is of an infectious nature, in that it is not always easy to prove that an infective material has not been introduced.

According to Anacker there is degeneration of the column of Goll, but no one else has been able to discover this lesion. According to Besnoit and Morel

there is severe parenchymatous inflammation of the smallest branches of the peripheral nerves, destruction of the processes of the axis cylinders and of the nerve sheath, while the nerve trunks and the spinal roots are uninjured. A more or less extensive chromatolysis of individual motor nerve cells was the only lesion that could be demonstrated in the cord. Cassirer on the other hand was unable to find any lesions in either the central or peripheral nervous system, and is consequently inclined to classify the condition as a functional nervous disease and connect it with Westphal's "pseudosclerosis," a disease of the human subject.

No exact knowledge as to the nature of the disease is available at the present time owing to the variable or negative results of experimental investigations. Taking the general nature of the disease into consideration it appears to be most likely that it is a polyneuritis. The negative results of the careful investigations of Cassirer are not compatible with this supposition.

**Symptoms.** The most striking symptoms in the early stages are, as a rule, excitability and fright. The animals have a scared look and the approach of a person or animal is sufficient to so frighten them that they stand and tremble. The appearance of a dog may cause collapse and epileptiform seizures. If a hold be taken of an animal many of the muscles are thrown into a state of spasmodic contraction. It is especially the muscles of the neck that are so affected, the head being drawn back in consequence. On warm days in summer the ears hang down and tremble, there is nodding of the head, and there may be dilatation of the pupils and nystagmus. The animals appear dull and depressed to a certain extent.

Gradually motor disturbances develop. Within one or two months from the onset of the disease, and it is usually earlier in warm than in cold weather, there is weakness of the quarters, the gait becomes uncertain and at the same time peculiar trotting-like movements are seen. The neck is extended and the head dropped, the hind feet are separated widely from each other, and very short, quick, tripping steps are taken, the joints being flexed to a very slight degree. In exceptional cases the feet are lifted very high and the animal walks like a fowl. Owing to the weakness of the quarters the animal cannot move quickly, much less jump over mounds or ditches. In the later stages the fore limbs become weak, the animals stumble along, keep falling down, rising being accomplished with great difficulty. Pressure on the back causes it to sink. If an animal be raised and set on its feet the joints are flexed or it falls on its knees. Defecation and urination are normal up to the end. According to Cassirer the reflexes and sensibility to electrical stimuli are normal.

In the majority of cases paresthesia and severe pruritus are present. In the early stages the animals gnaw at the root of the tail, the gluteal region and croup, and later the hind and fore legs, or they rub these parts of the body against the wall. In some cases they sit like dogs so as to be able to reach the irritating parts better with their teeth. The bare patches produced by the rubbing and gnawing become inflamed, the skin being reddened and thickened and covered with thick crusts. According to Funke a scratching or pricking of the affected portions of skin causes pronounced reddening.

In spite of the fact that the appetite remains good up to the end, emaciation, anemia, or hydremia, becomes more and more pronounced. Finally the weakness of the hind quarters becomes so great that the animals lie on the ground as if paralyzed, and emit hoarse groans. There is an offensive discharge from the nose and mouth, but they still gnaw places that they can reach. Finally they die from complete exhaustion.

**Course and Prognosis.** In the great majority of cases the onset of the disease is insidious and its progress slow. There may be apparent improvements from time to time, but the disease terminates fatally in two to four months. In very occasional cases the animals are in a condition of utter prostration within four to six weeks. The course of the disease tends to be more rapid in summer than in winter, and in young animals than in adults.

Recovery is very exceptional and when it occurs it is in the early stages of the disease.

The disease spreads very slowly, but in some cases the spread has been so rapid that in the course of a few years the profits have been out-balanced by the losses sustained, so that continued occupation of the farm becomes impossible.

Very occasionally there is a sudden outbreak of the disease on a farm previously free and within a short period great losses may be experienced, especially among the young lambs.

**Diagnosis.** Timidity, motor disturbances and especially the peculiar gait, and the intense pruritus combine to form a characteristic collection of symptoms.

The cardinal symptoms mentioned are not presented by every case, but since a number of animals are always affected at the same time the incomplete series of symptoms presented by any individual animal can often be completed by examination of other animals. Diagnosis is most difficult where only a few animals are affected, as in the case of newly purchased rams, and when the symptoms presented by such cases are not characteristic. More extensive observations will furnish evidence as to the nature of the disease.

The disease may be distinguished from other diseases characterized by pruritus, and in particular from scab, by the fact that the skin appears to be healthy save for the lesions caused by the rubbing. In other diseases no motor disturbances are observed. Cœnurosis has often been mistaken for this disease, the development of cysts in the vertebral canal sometimes causing sacral paralysis, but in cœnurosis there is never any timidity, trembling or pruritus.

**Treatment.** Up to the present no satisfactory treatment has been devised, and the animals should be slaughtered as early as possible.



**Prophylaxis.** A rational plan of breeding must be adopted and all pampering of the animals must be avoided. The greatest care must be exercised in the matter of the purchase of fresh animals as these may be the source of infection to the whole flock.

After symptoms have made their appearance it is a matter of the greatest difficulty to check the spread of the disease. The affected sheep and those that have been in immediate contact should be isolated, and the remainder of the flock should be removed to a high-lying district.

**Literature:** Besnoit, *Rev. Vét.*, 1898, 397.—Besnoit & Morel, *ibid.*, 1899, 265.—Cassirer, *V. A.*, 1898, CLIII, 1.

**Occurrence.** Attacks of vertigo are chiefly noticed in horses and dogs. Generally it is, with very few exceptions, that draught horses fall ill, whereas blooded horses and those of the hardy country type are seldom afflicted.

**Etiology.** In the domesticated animals megrims scarcely ever occurs as an independent affection (*vertigo idiopathica s. essentialis*), but as a rule as a secondary ailment (*vertigo symptomatica*).

Brain diseases may above all form the basis of megrims. Hyperemia, hydrocephalus, tumors, parasites, contusion and hemorrhage into the brain, emboli of the cerebral vessels, inflammation of the brain and its coverings come under this category in about equal proportions. Also diseases of the cerebellum and its adjoining parts of the brain are, in all probability, often accompanied by true vertigo.

Sometimes vertigo is associated with defects of vision. Observations of attacks of megrims through disturbance of the motor power of the eye muscles have not been recorded but it is certain that suitable external irritation of the organs of vision can produce attacks of staggers, as many observations prove. Thus horses are at times attacked with vertigo when travelling on unequally lighted highways, between two rows of trees, or when moving quickly under a rising or setting sun, when running round in a circle for a long time, or when objects move quickly before their eyes as in railway journeys. Besides many authors blame the glistening inner surface of the blinkers which reflect rays of light for causing attacks of vertigo.

Of diseases of the organ of hearing, affections of the labyrinth or of the *n. vestibularis* may occasion megrims (compare Menière's disease).

Disturbances of circulation may result in vertigo (*n. cardiaca*) such as every form of heart weakness, compression of the veins, pericarditis, growths inside the pericardium or in the neighborhood of the base of the heart. In this connection, compression of the superficial veins in otherwise healthy horses may be noted; this may be due to parts of the harness (the collar or throat latch) or to the head being pulled and held in (in draught horses). Far more readily may cerebral anemia cause attacks of megrims.

Of the diseases of the digestive organs, intestinal catarrh or helminthiasis (*v. verminosa*) sometimes lead to attacks of dizziness.

Violent irritation of the skin may occasion an attack especially if occurring on the injured skin; Guibert has seen an attack of vertigo after simply brushing over a short clipped fetlock.

Finally megrims occurs as a symptom of acute poisoning and indeed as a result of a direct effect of the poison on the central nervous system. Such an effect is produced by alcohol

and by the other narcotics, as also by some poisonous plants (*Solanum*, *Equisetum*, *Lolium temulentum* and reed, *Phragmites communis*, etc.).

**Symptoms.** Horses are attacked chiefly when at work. The animal suddenly manifests a staggering, slackened or uncertain gait or it suddenly stops, nodding or shaking the head, snorting conspicuously, pulls back in its harness, leaning against the pole or the neighboring horse, after stopping they spread the feet apart, the head is held sideways, the horse sways in the direction of the inclined head, or presses with uplifted head backwards or with sunken head forwards. These disturbances of equilibrium sometimes last only a few seconds and the attack is soon over so that after a short time the animal can continue its work. In other cases the animal completely loses its equilibrium, loss of consciousness occurs and often also nystagmus, the animal collapses, falls (*vertigo caduca*), and remains for a long time lying quietly, then it commences to kick, suddenly stands up, shakes itself and appears all right again. If the attack occurs in the stable, the horse staggers to and fro with outspread limbs, holds its head up or to one side, hangs on the halter strap, or leans against the partition or wall and suddenly falls to the ground. The course of vertigo runs similarly in the other species of animals.

At the beginning of an attack the animal shows great anxiety, its gaze is fixed, the pupils are dilated, respirations are quickened and sweating is general all over the body. Dogs frequently vomit and at times urinate and defecate involuntarily.

Individual attacks last barely for one or two, exceptionally up to five minutes, but recur at varying intervals of time. Horses that are subject to vertigo are most frequently attacked during work and in warm weather, whilst in winter they are seldom affected. When being led or ridden, horses subject to vertigo are seldom attacked.

The name *vertigo abdominalis* was formerly applied to a disease peculiar to horses. Its symptoms on the one hand were severe digestive disturbance (diminished appetite, colic, tympanitis) and on the other great dullness and possibly also forced movements and great excitement. Such attacks were repeated, and finally after some hours or days the patient recovered or at times died. It is very probable that this affection was due to poisoning by plants.

Seasickness causing a dizzy feeling is occasionally seen in animals. Damoiseau saw it in an Arab horse transported from Syria to France. On the first day, the animal was depressed, alternately bending and stretching his head, neighing often, trembling all over his body and vomiting after each feed for four days; in eight days he had recovered. Hering has repeatedly seen the same illness in dogs.

**Diagnosis.** Vertigo may at all times be mistaken for epilepsy; it is differentiated, however, from this affection by the absence of convulsions. Restiveness and fright are scarcely noticeable since in these the disturbances of equilibrium are



absent. The cause of vertigo is hard to establish and its discovery is especially necessary in newly bought horses.

**Treatment.** At the beginning of an attack of megrims the horse should immediately be unharnessed and by supporting the head and shoulders the animal is prevented from collapsing. Covering up the eyes, and cold douches to the head, also exercise a favorable influence. If the fall cannot be avoided, one must at least take care that the animal does not injure itself. After the passing of an attack, a judicious rubbing of the body and limbs has a good effect.

The treatment is directed to the cause of the vertigo and if this can be ascertained complete recovery may be expected. By judicious care and cautious use of the animal, by removal of the disturbing blinkers, as well as by the employment of proper harness, the best results will be attained.

**Literature.** Dexler, *Nervenkrkh. d. Pferdes*, 1889, 231.—Guibert, *Rec.*, 1899, 725.—Hering, *Spez. Path.*, 1858, 658.—Lustig, *D. Z. f. Tm.*, 1878, IV, 17.

**Menière's Disease.** (*Vertigo ab aure laesa*, V. labyrinthina.) By this is understood in human medicine, vertigo, caused by a neurosis of the nerves of hearing and which is accompanied by strong buzzing in the ears. The affection is often caused by disease of the arched passages of the membranous labyrinth which is well known to play an important part in the preservation of equilibrium.

A similar morbid condition was noticed by Fleming in several horses with the following symptoms: The animal suddenly shook one ear, bent its head to the same side, moved in a circle or pushed to one side, had a fixed gaze and finally fell on the side on which it shook its ear; after a time it stood up but remained excited for a long time.—Similar cases have been noticed in horses by Fröhner, and Lellmann saw one in a cat which afterwards became deaf.—(Fleming, *The Vet.*, 1891, 466.—Lellmann, *B. t. W.*, 1902, 776.)

In chicken pest similar attacks of vertigo (twisting of the head, nodding of the head, circular movements, rolling) have been noticed in hens (Centanni, Ostertag & Wolffhügel), and oftener in geese, further in artificially infected young pigeons (Vol. I). The authors saw similar symptoms in two hens affected with purulent otitis or purulent inflammation of the petrous temporal bone.

**Paralytic Vertigo.** (*Vertigo paralysans*.) Gerlier noticed in the Canton of Geneva among laborers and herdsmen repeated peculiar attacks of vertigo with passing paralysis of different muscles, occurring as an epidemic. He also saw similar attacks in the same neighborhood in several cats. The symptoms were as follows: the animal stopped suddenly, shook its head often to one side, and finally bent it until its nose touched the ground. In severe cases, the animal fell on its belly, and remained a few minutes lying motionless on the ground. After the passing of the attack the animal stood up again and moved about in a normal way. Many times on account of the relaxation of the muscles drawing down the lower jaw pseudotrisismus was observed. During the attacks the animal did not see although consciousness persisted. The attacks were

influence on the rump and the hind quarters, an epileptic attack in the domestic animals must arise chiefly in the cortex of the brain, passing over the motor centers by way of the extra pyramidal tracts or in these latter structures themselves.

In dogs, faradisation of definite areas of the cortex causes spasms which are quite like typical epileptic attacks. The spasms at first occur in the parts of the body which are supplied from the stimulated areas and the remaining portions of the body may be affected with spasms in the same succession in which the corresponding motor regions in the cortex follow one another. The attack also occurs if complete parts of the cranial cortical substance up to the motor zone have been removed, but remains absent if only the motor region is extirpated, the other cortical section being retained. After partial destruction of the motor cortical zone one may succeed in occasioning an attack but the muscles of the parts of the body whose corresponding centers have been removed take no more part in the spasmodic contractions.

The stimulation of the sub-cortical centers, after removing the motor region of the cortex, causes general convulsions and irregular spasms (Ziehen, Binswanger, Prus).

Up to the present time the nature of the causes of epilepsy are not known exactly. It is assumed that certain parts of the brain possess a greater degree of irritability than normally or that their molecules themselves are in a state of very unstable equilibrium, being affected easily and markedly by insignificant irritation such as variations of blood pressure which produce no effect whatever on the healthy brain. Of the nature of this increased irritability no information is at hand, it has been only observed that the ailment itself and the predisposition to it may be transmitted to the progeny. In the individual with an hereditary predisposition the disease can occur at a certain age without traceable cause, but more frequently it happens that the first attack is due to some external influence whilst later attacks may arise without any external cause. Such occasional causes appear to be pretty frequent in animals, for instance severe psychic disturbances (fright, excitement, punishment), dazzling rays of light (Lövy produced several attacks a day in a cow from this cause) less frequently shocks to the body, blows, falling down, etc.

In more recent times it is often assumed that epilepsy may be due to an auto-intoxication. In the many experiments conducted on men for the determination of definite poisons in the metabolism of the epileptic, the experiments undertaken by Krainski may be mentioned especially. According to these investigations epilepsy is produced by the formation of larger amounts of ammonium carbamate in consequence of an anomaly in metabolism, which then irritates the cortex cerebri.

Spasmodic attacks similar to epilepsy have also been produced by Hahn, Massen, Nencki and Pawlow in dogs with carbamate of calcium and sodium, but Putnam & Pfaff could not confirm the findings of Krainski. In two cases Donath is inclined to ascribe epileptic convulsions to cholin.

Heredity is commonly looked upon as a cause of the predisposition. There are trustworthy records showing that the af-

fection can be transmitted by the father or mother animal to the offspring.

Of observations in this connection the following may be mentioned: Reynal noticed the hereditary transmission of epilepsy in three succeeding litters of an affected cat. La Notte saw the disease in two herds in the descendants of two affected bulls, and Cruzel in three calves from an epileptic cow. In a case observed by Otto a bitch transmitted the complaint to two of her progeny.

The epileptoid attacks known under the name of **secondary or reflex epilepsy** differ from true epilepsy in that they are the result of disease of the brain or of other organic diseases or pathological conditions, after the removal of which they disappear, leading neither to a disposition to further attacks in the sick animal nor being transmitted by heredity to the progeny.

Epilepsy-like attacks arise in such diseases of the meninges or of the bones of the skull as are capable of causing, for a time, an acute excitation in the corresponding parts of the brain. This excitation may be confined to the immediate neighborhood of the active cause, such as an exostosis or splinter of bone, and the attacks of convulsions are therefore limited to certain parts of the body (Jackson's Epilepsy). Such epileptoid attacks will generally be due to traumatic causes which produce a partial depression of the roof of the skull, to the pressure of a splinter of bone into the cortex of the brain or to the occurrence of hemorrhage between the meninges of the brain and the brain itself. In guinea pigs epileptoid attacks have been produced artificially by traumatic influences (blows on the skull) (Westphal). Further causes are solitary tubercle seen particularly in the brains of swine and cattle, parasites (especially *Cysticerci*) in the brain, and in the pia mater of swine, perhaps also in dogs, much more rarely neoplasms, abscesses, exostoses on the internal surface of the cranial bones, ossifying inflammation of the dura cerebri, etc., old encephalitic foci lead, especially in dogs, to epileptoid attacks (Dexler). In all these cases the first attack is usually induced by the same immediate causes as in genuine epilepsy.

Reflex epilepsy is mostly associated with certain diseases of the mucous membranes. Thus epileptoid spasms in dogs very often result from the presence of intestinal worms (*Tæniæ*, especially *T. echinococcus*; *ascarides*) or even on simple constipation. Similar attacks may be caused in swine by the *Echinorhynchus gigas*, in cows by the *Tænia denticulata*, and in horses by the *Ascaris megalocéphala*.

The attacks in this kind of reflex epilepsy are usually not the result of a reflex effect produced by pain, but either by irritability of the vaso-motor nerves in the cranial cavity, or much more by the assimilation of metabolic products of the parasites or by the putrefaction caused in consequence of the disease of the bowel.

Epileptoid attacks have also been at times observed in severe disease of the external auditory canal as well as in the



presence of pentastomes in the nasal cavities. Chronic uremia may likewise produce epileptoid attacks. Much less frequently the irritation which produces the convulsion originates in wounds of the mucous membrane (sharp teeth!) or in painful wounds or cicatrices of the skin; in very exceptional cases the irritation of a perfectly healthy part of the body was found to produce convulsions (Gerlach, Schrader, Dörrwächter). In most cases, however, injuries of the withers, fractures of the horns, or in the dog injuries of the sole of the foot are responsible for attacks of reflex epilepsy.

Brown-Séquard produced reflex epilepsy in a guinea pig artificially by injuries to the n. ischiadicus, and the disease was claimed to be transmitted to the progeny, yet later authors (Sommer, Karplus) could not prove this in a single case.

**Anatomical Changes.** In true epilepsy no morbid changes can, as a rule, be shown on postmortem, either in the brain or its immediate neighborhood; nor does microscopic examination usually give any results, although it is probable that the nerve cells of the cortex or other elements of the brain are diseased; but the minute changes cannot be demonstrated by the available methods of investigation.

Since Siedamgrotzky noticed the illness in a dog with asymmetry of the skull, and Bassi the same thing in 9 out of 15 epileptic horses, since further Fabretti found the same condition in a cow and two dogs, this condition must in the future be looked for at autopsies.

**Symptoms.** In man certain prodromal symptoms precede the epileptic attack, which since the days of Galen have been known by the name of "aura epileptica" and these occur sometimes also in animals. Thus many horses appear sluggish before an attack, pay no attention to the word of command or become excited at it; others commence nodding and shaking the head or attempt to scratch it with the hind feet. Swine go about restlessly, root up the ground with the snout and grunt uneasily. Dogs are sometimes depressed, they run restlessly to and fro, toss the head and bite the nearest object. In the great majority of cases such preliminary symptoms are, however, wanting or escape notice.

The **severe epileptic attack** (grand mal) declares itself in the great majority of cases in the following symptoms: The animal suddenly exhibits great anxiety, stands with a fixed look and wide open pupils as if nailed to the ground, soon begins to tremble, breathes deeply with maximal opening of the nostrils, reels to and fro, seeks for a time to preserve the equilibrium by straddling out the limbs or by stepping forwards or backwards, but soon falls unconscious to the ground. Cattle at first often emit a peculiar hollow bellowing, dogs utter a loud shriek, whilst sheep make circular movements; dogs get up sometimes after collapsing, make a few reeling steps, but soon break down again.

haustion or muscular weakness. Holterbach noticed epileptic attacks in a cat which only occurred at night time (*E. nocturna*).

The attack may be of different degrees of severity and of varying duration. After collapse in horses there may be merely stiffness, lasting for a few seconds and during which all the vegetative functions appear to be extinguished, but soon evacuation of dung and urine occur and the attack comes to a conclusion. Certain symptoms may be absent, such as the increased secretion of saliva and the champing of the saliva into foam, and further involuntary voidance of dung and urine. Generally the muscles of both halves of the body will be attacked equally, but many a time the spasms are much more severe on one side of the body than the other. Horses, dogs and swine seem to have much more intense convulsions than cattle and sheep.

The sequence of the symptoms also does not always follow in the order described as above, inasmuch as the spasms sometimes begin at an extremity such as the toe, and then gradually attack the remaining parts of the body, extending finally to the head.

In birds the symptoms are in a great measure as follows: The bird emits sharp sounds, makes flapping movements with the wings, falls on its side or back, moves its feet quickly, rolls its eyes, bends the neck round to one side, alternately opens and shuts the beak and throws its whole body to and fro. After 1 to 2 minutes the spasms cease, whereupon the bird lies for a long time as though in a stupor and without sensation; afterwards it stands up, but still staggers about and supports itself with its beak and outstretched wings until it finally stands on its feet, or with regular flapping of the wings flies away (*Trasbot*).

The **slight epileptic attack** (*E. minor*, *petit mal*) is a rudiment of the epileptic seizure proper (*Weygandt*) and declares itself in various disturbances of consciousness while the symptoms of motor excitation occur at circumscribed areas for a short time only or are absent altogether. There are also records of similar conditions associated with local spasms, in which the animals did not break down when they could lean against a supporting object. It is, however, very difficult to recognize, and distinguish between such cases of secondary attacks of vertigo and convulsions in animals.

Of the psychic abnormalities noticed in human patients suffering from epilepsy, and occurring also in the intervals between the attacks, one sees nothing in animals. In dogs intelligence and attentiveness have suffered according to many authors (*Gohier*, *Reynal*) yet it does not appear certain that these were secondary epileptic attacks, but more likely due to encephalitis following distemper.

**Secondary epilepsy** or the **epileptoid attack** agrees in its external form with true epilepsy, only the symptoms may usually be ascertained as due to organic disease of the brain or to other diseased condition.

Secondary epilepsy occurs at times as so-called Jackson's

or cortical epilepsy, in which besides loss of consciousness the spasms always affect the same group of muscles, and then proceed to attack the other muscles successively.

**Course.** True epilepsy is a chronic affection that causes a continued predisposition to periodically recurring attacks of convulsions. The attacks come on, however, at different intervals; in many animals few attacks are noticed during the whole course of life, but in others seizures may occur weekly, daily or several times a day. Their appearance sometimes seems to depend on external influences, and again they follow in short periods without any apparent cause. It seems as if at first the attacks were less frequent but longer, and later more frequent but of shorter duration. In the most severe cases the attacks follow each other quickly and the condition of the animal becomes such that consciousness does not return in the intervals (*status epilepticus*); in such cases the convulsions themselves can cause death, whilst ordinarily severe epilepsy may cause the animal to injure itself fatally when collapsing. In some animals the attacks are at times very frequent, then they subside for a long time, even for several months, whereupon several attacks occur at short intervals and then disappear for a longer period.

Whether true epilepsy in animals is curable, can not be decided by the clinical observations, but a predisposition for further attacks of convulsions appears to persist invariably.

The course of secondary epilepsy depends on the underlying disease.

**Diagnosis.** For the recognition of genuine epilepsy the establishment of the chronicity of the affection appears to be of primary importance, but due weight must be given to whether during the periods between the attack the animal exhibits any organic disease of the brain or other diseased condition which stand in causal relation to them. If investigation in this respect leads to a negative result, but the attacks of spasms are always accompanied by loss of consciousness, the nature of the complaint is not hard to recognize. Limited or general tonic-clonic contractions have been observed in acute diseases of the brain, in many infectious affections (*distemper*), also in certain cases of poisoning. But in these cases symptoms of the underlying disease are present also in the intervals between convulsions, which are usually short, and moreover the diseases always take an acute course. The last mentioned condition differentiates also the so-called eclampsia of the bitch from epilepsy, and moreover the close connection of eclampsia with parturition will reveal the nature of the complaint.

It is much more difficult to differentiate in a given case between true epilepsy and an epileptoid attack (secondary and reflex epilepsy); but since prognosis as well as the course of treatment will depend on a clear understanding of this point,



one always must carefully investigate the signs of illness which might underlie the attacks. Only if by continued observance such cannot be proved will the assumption of a distinct epilepsy be justified, and one must always reckon with the possibility that this hypothesis may be upset at the postmortem. Hereditary taint and long intervals between the attacks generally denote a true epilepsy.

**Prognosis.** In true epilepsy this is unfavorable since a permanent cessation of the attacks can scarcely be expected. The influence of the ailment on the usefulness of the animal varies very much in different cases. In this respect an unfavorable view must be taken, especially if the attacks occur at short periods. The hereditary affection generally takes an unfavorable course, and more especially so if the first attacks occur in early age.

The consideration of secondary epilepsy depends upon the nature of the causative illness; if this is curable there is hope of complete recovery from the attacks. On this account the prognosis of secondary epilepsy is generally more favorable than that of genuine epilepsy.

**Treatment.** Of the numerous methods of treatment in true epilepsy the internal administration of bromides has proven most successful if these drugs are used systematically in proportionately large doses and for a long time. With this object potassium bromide may be given to dogs in daily doses of 3 to 4 gm., sheep and swine 5 to 6 gm., large animals 20 to 50 gm. (in freshly prepared electuary or solution!) for 2 to 4 weeks. If during the pause renewed convulsions occur the course is to be repeated. The other salts of bromine may also be employed, and at times these seem to be of advantage; thus one can order potassium, sodium and ammonium bromide together in the same doses as the potassium bromide alone, in which case one daily dose or at most two are to be given. For birds 1% to 5% potassium bromide solution as drinking water is the best. The much more expensive bromipin may be given to dogs in doses of one tea or table spoonful. If signs of a chronic bromine poisoning (emaciation, trembling, gastric catarrh, and especially eczema or acne in different parts of the body) occur, then the treatment must be suspended for a time, and mild aperients combined with arsenic must be used.

Further, one must give suitable nourishment (if possible food containing little chlorine and nitrogen) assuring a proper digestion, and also exercise care that the animal does not come in contact with any influences that bring on the attack. Considering the probability of the hereditary nature of the illness breeding animals must be excluded from breeding (according to Röhl the attacks sometimes cease after castration), working animals must be employed cautiously.



During the individual attacks one must protect the patient from injury. But attempts to abort the attack are scarcely successful; the procedure recommended for this purpose (passive movements of parts of the body in opposite directions, wetting the body with cold water, etc.) usually produce no effect. Only if the convulsions follow one another very quickly, almost continuously, one should seek to allay the irritability of the cortex of the brain by inhalations of chloroform or ether or by injections of morphine.

The treatment of secondary epilepsy is to be directed against the underlying illness. In so far as this is curable at all, the employment of narcotic remedies and the suitable treatment (expulsion of worms, artificial removal of gadfly larvæ or pentastomes, relief of existing constipation, excision of cicatrices, etc.) must be undertaken. Otherwise the treatment of epileptoid spasms in organic diseases of the brain is usually similar to that of true epilepsy. In Jackson's epilepsy operative treatment seems to be indicated; the favorable experiences made in human medicine in this direction deserve at least proper notice. The necessity of operation is to be considered particularly in injuries to the skull, as well as where lesions are localized in the cortex of the cerebrum.

**Literature.** Bassi, Pastore, *Mod. Zootriario*, 1904, 201.—Dräseke, *Psych.-Neurol. Wochenschr.*, 1906, 1.—Dexler, *Nervenkrkh. des Pferdes*, 1899, 224 (Lit).—Fabretti, *Rec.*, 1904, 516.—Holterbach, *B. t. W.*, 1908, 656.—Kramell, *Z. f. Vk.*, 1905, 498.—Lövy, *Vet.*, 1892, 446.—v. Monakow, *Gehirnpathologie* 1905, 1, 248 (Lit. on experiments in brain irritation).—Otto, *S. B.*, 1902, 70.—Sarbo, *Die Epilepsie*, 1904, (Hungarian).—Schröder, *Z. f. Vk.* 1902, 25.—Sommer, *Neur. Cbl.*, 1901, 152.

### 3. Eclampsia.

By the name of eclampsia convulsions are designated which are in every respect similar to those of epilepsy, but which either occur only once or run an acute course with several recurrences. True eclampsia includes only those attacks which occur independently of organic diseases, that is which occur independently as pure neuroses.

Eclampsia corresponding to this definition ordinarily occurs only in very young dogs and pigs and is rarely observed in older animals.

The **eclampsia of young animals** occurs almost exclusively at the time of teething. Many authors are therefore inclined to consider that there is a causal connection between these attacks and the increased sensitiveness of the gums when teething; but in many cases some disease of an internal organ may be shown to exist, such as intestinal worms, acute intestinal catarrh, inflammation of the mucous membrane of the mouth, infectious diseases, etc. Poorly developed and particularly rickety animals appear especially predisposed to the complaint.

The eclamptic attack manifests itself mostly by quite simi-

lar symptoms as does the epileptic. Here also certain warning signs may be noticed such as unrest, aimless wandering about, loss of appetite, starting up from sleep in a fright. Sometimes the attack lasts only for a short time and then passes off for good, but again it may last several quarters of an hour or be repeated at different intervals while in other cases the attacks may follow so quickly on one another that the animals are continually tormented by the convulsions for hours (status eclampticus).

Eclamptic attacks quite frequently lead to death, and this may happen in a few minutes. As a rule the prognosis is more unfavorable the longer the convulsions continue. In giving an opinion caution is always necessary in order not to mistake other ailments for eclampsia. Thus dogs in the course of distemper, swine in erysipelas or pyobacillosis not infrequently exhibit convulsions; besides the spasms of the death struggle are sometimes believed to be an independent disease by the attendants.

Since eclampsia of young animals is in many cases a reflex condition, treatment consists in the removal of the cause if this has been ascertained, and the driving out of tapeworms is here of first importance. The attack itself is controlled with narcotic remedies, such as potassium bromide (0.2 to 0.5 gm. every 3 hours), bromipin (a teaspoonful to a tablespoonful) or with chloral hydrate (0.1 to 0.5 gm.).

The rare **eclampsia of adult animals** is most apt to be due to the transitory effect of the same causes which bring about reflex epilepsy (see page 771). Tapken saw in a cow an attack of convulsions lasting for several hours due to the noise made by the firing of a gun.

White & Plaskett report upon a North American breed of goats, ("Startled goats," "Fainting goats") where the animals collapse from quite trifling external influences, and are seized with tonic spasms like those from strychnine poisoning; after 20 to 30 seconds they get up, and their gait remains stiff for a short time. Many animals are said to be frightened to death from quite trivial causes (!). According to Dexler these cases are due to pathological and degenerative peculiarities of an hereditary nature similar to the sensory disturbances in dancing mice and albinotic deaf animals.

**Literature.** Besnoit, Rev. vét., 1901, 349.—Castelet, D. t. W., 1900, 312 (Rev.).—Dexler, B. t. W., 1908, 970.—Soulet, Rev. gén., 1905, VI, 471.—Tapken, D. t. W., 1899, 353.—White & Plaskett, Am. v. Rev., 1904, 1167.

#### 4. Puerperal Convulsions.

##### (*Eclampsia puerperalis*.)

In connection with parturition there occurs a peculiar convulsive illness chiefly noticed in bitches, but occasionally in other female animals. In the course of an attack consciousness does not, as a rule, appear to be visibly disturbed. The last men-



tioned circumstance and the fact that the trouble is associated with parturition distinguishes the affection both from epilepsy and eclampsia, and appears to justify its separate treatment.

(a) Puerperal Convulsions in Bitches.

(So-called *Eclampsia of Bitches.*)

**Etiology.** The affection, about the character of which nothing is known, is noticed most frequently in pampered pure bred bitches.

The localization of the irritation which produces the spasms may be in the cerebral cortex, in the subcortical motor centers, or in the anterior horns of the spinal cord, but as consciousness is undisturbed until the end it is probable that the two last structures are concerned most often. In view of the fact that similar convulsions are observed in strychnine poisoning and in tetanus it is not out of the way to assume that the spasms are due to the direct effect of some toxins which are formed somewhere in the body, possibly in the organs of generation, and act on the motor cells of the anterior horns of the spinal cord. This view is more credible than the hypothesis of Friedberger & Fröhner, who believe that the immediate cause is to be found in a peculiar reflex irritation arising in the udder or uterus and bringing about anemia of certain centers of the brain. Reul sees a certain etiological similarity between this disease and parturient paresis. It cannot be wholly rejected that the affection represents, as do puerperal convulsions in general, an anaphylactic phenomenon brought about in the course of pregnancy by the homogenous, specific placental albumen, so that after the absorption of considerable quantities of placental tissue anaphylactic symptoms arise. The eclampsia of lying-in women is nowadays considered in many cases in the nature of anaphylactic shock.

Vassale produced eclamptic attacks in three pregnant bitches by extirpation of the parathyroid glands, and these could be brought to a standstill by the administration of large doses of parathyroidin. On this account he assumes that in a latent parathyroidal insufficiency in woman in the last third of pregnancy severe convulsions occur.

In the great majority of cases the illness arises during the first few days after parturition, exceptionally, however, after a longer time, or it may be before the birth in the last stage of pregnancy. Friedberger saw a case 50 days after birth, Duet 8 days before, Albrecht 63 days after oestrus. According to Uebele the illness may occur in bitches which are not pregnant and not suckling, at the time where in case of conception parturition might have been expected and lactation has also been known to occur at this time. Otherwise the occurrence of convulsions are believed to be dependent on certain accidental causes producing violent mental disturbances (sorrow over lost young, fright, fear of punishment), catching cold, etc.; but such incidents can just as often not be shown to have occurred. Animals once ill are inclined to be attacked at subsequent parturitions (Gajewski).

The influence of excitement after taking away of the young is generally exaggerated; thus in a third of the cases observed by Müller no young had been taken away from the affected animals.

**Symptoms.** The illness commences, as a rule, shortly after normal and easy birth (only exceptionally earlier than after 48 hours). The animal becomes restless, whimpers and often

cries out continuously, its look is anxious, the breathing quickened and labored, the mucous membranes appear greatly injected. With a rapid aggravation of these symptoms and a rise in temperature the gait becomes staggering after 10 to 15 minutes, and soon the animal is incapable of standing on its feet, falling on one side and stretching all four feet out with such a force that it is possible to bend the joints only by the employment of great force. From time to time one sees also fibrillary twitchings or even strong muscular contractions which, spreading towards the head, end in general convulsions followed by muscular rigidity.

During the whole duration of the illness consciousness appears to be retained, the animals respond to calls and are at times even excited so as to snap at the hand. In look and expression they exhibit great anxiety, their pupils appear to be normal in size and capable of reacting (in exceptional cases they are said to be dilated and fixed). Breathing is much quickened and strikingly labored, the animal panting with the mouth open and tongue protruding; but from time to time chewing and swallowing movements occur, and thus the saliva which has accumulated in the mouth is swallowed while otherwise it flows out from the corners of the mouth. The pulse feels small and hard, the mucous membranes are cyanotic. The milk glands appear at first tense and warm, although later they become flaccid; they do not secrete any milk from the start.

The spasms sometimes continue only a few hours, but usually last 1 to 3 days. According to several authors spontaneous cure only occurs exceptionally, but the authors cannot confirm this.

**Treatment.** This consists in the employment of narcotic remedies, and almost always yields favorable results. The simplest and most suitable appears to be the subcutaneous injection of 0.02 to 0.05 gm. of morphine, whereupon the convulsions promptly decline and finally disappear; if no improvement occurs in 1 or 2 hours the injection may be repeated. Of good effect are also the other sedative and narcotic drugs, such as chloroform (cautious inhalation up to commencing narcosis), chloral hydrate (0.2 to 5.0 gm.), urethan (5 to 20 gm.), hypnon ( $\frac{1}{2}$  to 2 gm.), veronal ( $\frac{1}{2}$  to 2 gm.), Zündel's chloroform syrup (1:100, a teaspoonful every  $\frac{1}{4}$  hour, later every 2 hours), etc. Occasionally parathyroidin may be tried.

**Literature.** Albrecht, W. f. Tk., 1901, 469.—Friedberger, Münch. Jhb., 1876-77, 103.—Gajewski, A. f. Vet.—Wiss., 1882, 145.—Massoglia & Sparapani, Rev. gén., 1907, X, 495 (Rev.).—Vassale, Vet. Jhb., 1906, 149.

#### (b) Puerperal Convulsions in the Other Animals.

In **horses** the disease has been observed once by Tapken. In a mare of somewhat advanced age which after earlier births had shown slight spasms, an attack lasting five minutes came on two hours after a difficult



parturition. It manifested itself by clonic spasms of the muscles of the head and neck and of one hind extremity, as well as by shallow breathing. On the following days a few attacks were noticed, but they disappeared after removal of the afterbirth.

In **cows** Saint-Cyr & Violet, Flemming, Albrecht and others have noticed attacks shortly after parturition which were manifested by clonic spasms of the muscles of the trunk and extremities, by rotation of the neck and grinding of the teeth, and were usually repeated several times lasting as long as 2 to 3 hours; with the exception of one case, in which intermeningeal hemorrhage occurred with fatal result, they all ended in recovery.

In **sows** Tapken often saw attacks of convulsions in connection with parturition; these occurred even during parturition, which was usually severe, less often immediately before or shortly after the act. The whole body of the animals which at the time of parturition were lying on the side, exhibited extensive muscular twitchings, there was rolling of the eyes and spasms of the muscles of mastication. The individual attacks lasted from  $\frac{1}{2}$  to 1, seldom 2 to 4 minutes, and were repeated at short intervals. The convulsions recurred very frequently, and often rendered slaughter of the animals necessary. Similar cases were observed by Hengen, Giovanoli, Holterbach and Sonnenberg. The last named author found fatty degeneration in the parenchymatous organs and tube casts in the urine; he considers the disease as the result of an auto-intoxication corresponding to the puerperal eclampsia of women.

The causes of the convulsions are quite unknown. Although one cannot exclude from consideration an irritation of the nerves in the generative organs during parturition, yet such a conjecture is not suitable for most cases, because attacks occur after easy births, and sometimes indeed a few days after or before the act. A more probable conception is that the disease is produced by toxic material formed in the body or perhaps in the generative organs.

The treatment consists in the employment of narcotics (Hengen caused cessation of parturient convulsions in a sow by giving 10 grammes of chloral hydrate in electuary).

**Literature.** Albrecht, W. f. Tk., 1880-89.—Giovanoli, J. Vét., 1905, 283. (Rev.).—Hengen, W. f., Tk., 1900, 365.—Holterbach, B. t. W., 1905, 832.—Sonnenberg, *ibid.*, 1906, 945.—Tapken, D. t. W., 1899, 353.

### 5. Parturient Paresis. Paresis puerperalis.

(*Milchfieber, paralytisches Kalbefieber, nervöse oder toxische Form des Gebärfiebers* [German]; *coma puerpérale, fièvre vitulaire* [French]; *parturition fever, milk fever, calving fever* [English].)

Parturient paresis is an afebrile disease which occurs at the termination of parturition and which is manifested by sudden loss of consciousness and a generalized paretic condition.

**History.** The disease was first described by German veterinary surgeons at the beginning of the nineteenth century, being first mentioned especially by Jörg (1808). The time when the disease became known corresponds with the period when it became customary to feed milk cows



more generously with the object of increasing the milk production. Together with the popularization of the intensive feeding of cows, the cases of disease also increased, and in a like measure from the middle of the last century the number of publications (see etiology) on the subject by veterinary surgeons in all the civilized countries increased also, without however clearing up the etiology of the disease. In the year 1897 the Danish veterinary surgeon, Schmidt of Kolding published a new theory as to the etiology of milk fever, which did not clear up the cause of the disease, although the treatment based upon it undoubtedly led to a very decided decrease in the losses arising from it. Recently Hess, Sonnenberg and Delmer have made experiments with the object of clearing up the etiology of the complaint, which is the only correct procedure.

**Occurrence.** Milk fever seems to be a disease almost exclusively confined to cattle, although a few authors (Saint-Cyr, Friedberger & Fröhner, Haubner & Siedamgrotzky, Hoopen) assert that it occurs, rarely, also in goats, sheep and swine. In certain regions and on individual estates the illness is very frequent and stands in close relation to the constantly increasing intensity of the agricultural operations (Hess); it is consequently to be considered as a disease of civilization. Accordingly it is met with much more frequently and in a more severe form on farms with choice, succulent food and with first class milk cows, also more often on the outskirts of towns than in distant localities or in the hill country (Hess, Knüsel).

**Etiology.** The actual cause of the disease is unknown. This much only is certain that in a great number of cases the influence of certain predisposing factors is manifest in a remarkable manner.

The breed of the animals has an influence inasmuch as cows of the Holstein breed, which give much milk, are predisposed to the disease somewhat less than those of the mountain breeds, and least of all are the range cows, although experiences in Hungary show that the range cattle are not entirely immune from the disease.

The influence of milk productivity is in so far evident that even in cows of the same breed the especially good milker is the one most prone to an attack.

An essentially predisposing rôle is played by the age of the cow, the great majority of attacks occurring in the fifth to tenth years of life, that is after the third to fifth calvings, while older cows and such as are under six years of age are attacked only exceptionally.

According to statistics collected by Jensen in Denmark out of 931 cases the age of the cows was: in 0.75% of cases 3 years; in 1.82%, 4 years; in 5.91%, 5 years; in 15.68%, 6 years; in 16%, 7 years; in 19.76%, 8 years; in 10.20%, 9 years; in 10.95%, 10 years; in 4.4%, 11 years; in 6.8%, 12 years; and in 7.62% more than 12 years.

The course of birth is of considerable significance since after easy births many more cases of illness are recorded than

after difficult ones (according to statistics collected in Bavaria out of 129 cases of illness 120 of the births were normal and easy), still cases also occur now and again after difficult parturitions. Hess further noticed that milk fever occurred only in such cows as went to full term or five to fifteen days beyond it.

A part that must not be undervalued is further played by the kind and mode of the feeding. Well-nourished cows chiefly become affected, especially those that are fed intensively towards the end of the period of gestation, as well as very greedy cows, especially those that have been dry for several weeks.

Most authors state that cows that are kept in the barn almost constantly are most susceptible to the disease, but Tapken could not determine any difference in this respect.

Finally some cows may be said to have an individual predisposition for attacks which occur at several subsequent parturitions (Röll).

Otherwise the illness arises in connection with the birth and indeed in most cases within the first three days, sometimes, however, somewhat later, or during or before parturition, yet never before the establishment of milk secretion (Hess, however, has never seen the onset of parturition paresis before or during birth in 170 carefully observed cases).

According to statistics collected by Jensen in 1107 cases, 4.24% occurred before birth and the remainder after birth; of the latter 2.53% began after 1 to 3 hours, 3.9% after 3 to 6 hours, 13.09% after 6 to 12 hours, 14.27% after 12 to 18 hours, 27.37% after 18 to 24 hours, 10.29% after 24 to 30 hours, 8.40% after 30 to 36 hours, 3.70% after 36 to 42 hours, 6.77% after 42 to 48 hours, and 5.42% after more than 48 hours. Hess did not observe any cases more than 96 hours after parturition, although there are records in literature where the disease was said to have occurred much later, as long as 10 days after parturition (Harms); but such cases may have been confounded with other diseases as is suggested by the investigations of Guillebeau and Hess.

The opinions as to the nature of the illness are numerous, but only hypothetical; some have no scientific foundation whatever, and are consequently untenable. Even those hypotheses which have more or less foundation do not throw a clear light on the etiology and nature of the disease.

Cerebral anemia is often assumed to be the cause of the symptoms of parturient paresis. This could arise from too powerful contractions of the uterus (Franck), or according to another view from an increased flow of blood to the udder, the effect of which would be accentuated by the milk secretion or the loss of tissue fluid caused thereby (hyposerohemia [Bredo]). In support of this hypothesis, on the one hand, the very favorable results of pumping air into the udder have been mentioned, and on the other hand also a few cases of rupture of the womb with hemorrhage, where symptoms similar to those of milk fever could be shown. It is believed by many that the udder and the blood vessels situated between the abdominal walls and the udder, are capable of containing about half the total quantity of blood in the body (Nelke, Keim).

The authors consider this hypothesis untenable for the following reasons: Marek did not succeed in producing milk-fever-like symptoms by very copious bleeding after calving. In one cow 30 hours after a normal calving 21%, and in another 36



hours after 27% of the total estimated weight of the blood of the body was taken away from the jugular vein in a space of time of from 20 to 70 minutes, without any disturbances being exhibited, except a moderately quickened pulse and pale color of the mucous membrane. The same was noticed in a heifer where 32% of the total quantity of blood was taken away. It was further noticed in two mares experimented on, where 42% to 44% of the whole quantity of blood was removed, that the only symptoms exhibited were quivering of the muscles and an anxious look. On the other hand Stahn in a case of fatal hemorrhage into the womb, without rupture, in a cow did not witness parturient paresis, the clinical picture of paresis puerperalis was only observed in such cows (Gebauer, Meier, Reinhardt, Habicht) where in consequence of uterine or vaginal rupture, not only blood but other matter passed into the abdominal cavity.

It is to be noted, moreover, that as long as the vasomotor apparatus is uninjured it always makes provision that the brain receive the needed amount of blood, not only in case of an increased flow of blood to other organs (with the exception of the very large total area of the abdominal vessels which are supplied by the splanchnic nerve), but also in case of a vasoconstriction in these organs. On the other hand experiments on dead animals can give no indication as to the total amount of blood in the udder of living animals. Watery solutions easily diffuse through the dead vessel walls into the adjoining udder tissue, while an injection of gypsum into the blood vessels, especially into the veins, causes an abnormally severe dilation of the vessels, since these exert an insuperable resistance in their fine branches. It appears, moreover, very strange that about half the amount of the total blood of the body should be present in the udder after parturition.

It would have to be ascertained whether or not in sudden changes the food contents of the abdominal cavity shortly after parturition a lowering of blood pressure occurs in such a manner that a decided diminution of the internal abdominal pressure causes a great determination of blood into the very wide vascular area of the abdominal organs for which even an uninjured vaso-motor apparatus cannot immediately procure efficient compensation.

Secondly, an **infection** is accepted as the cause of the disease. Supported by a certain similarity between parturient paresis and ptomaine poisoning, Zündel, Stockfleth and later also Schmidt-Mülheim assumed a toxic action, the toxins being due to a slowly progressing putrefaction of the lochia. This hypothesis was more or less related to that held by Eloire, Chauveau, Bissauge and Schneidemühl.—Nocard sought the cause of the disease in an infection proceeding from the womb with a resulting intoxication of the central nervous system, especially of the medulla oblongata, by toxins of staphylococci which increased in the uterus after parturition. In like manner Guillebeau & Hess assume an infection by streptococci and staphylococci through the genital tract injured during parturition; this would be similar to puerperal septicaemia, but with this difference, that in milk fever only an absorption of the bacterial poisons occur, which then result in paralysis of the vaso-motor centers, and through this a tense filling of the blood vessels of the abdomen, of the uterus, and especially of the udder itself.—The favorable result obtained by Knüsel with oxygen inflations of the udder led him to believe that there was an infection and intoxication proceeding from the udder and caused by anaerobic bacteria.

Against all these hypotheses many objections can be raised. First of all it has not yet been possible to demonstrate the supposed poison, putrefying lochia or the supposed pathogenic bacteria. (Streptococci, staphylococci and other bacteria can easily gain access to the uterus after parturition without necessarily producing at the same time a pathogenic effect.) Finally between parturition paresis and ptomaine poisoning (botulismus, allantiasis) there is an essential difference, for in milk fever the loss of consciousness is the principal symptom of the disease, whilst in ptomaine poisoning without particularly marked disturbances of consciousness paralysis is a



prominent symptom; besides milk fever although at first giving the impression of a very severe illness, generally ends in recovery, whilst ptomaine poisoning and other bacterial toxins generally lead to death if once they have caused a severe illness. Also in a general vasomotor paralysis it is hard to believe that there is a copious flow of blood to the udder, because in this condition the slackened blood vessels supplied by the splanchnic take up the greater part of the blood so that the other organs situated outside of the abdominal cavity, and consequently also the udder, experience a deficiency of blood. Finally the diminution of milk secretion as well as also the slackness and flaccidity of the udder that are observed frequently cannot be made to agree with a hyperemia of this organ.

Recently many veterinary surgeons have been inclined to accept the hypothesis of the existence of an autointoxication. Ehrhardt and Albrecht held the view that protein substances collected in the tissues of the mother animal and used up by the fetus, during pregnancy, may after birth be changed into material having a toxic effect. Eber's view was that a toxigenous substance originated in the pregnant uterus which was changed into a poison by the active metabolism in well nourished vigorous cows, whilst Mamadysecky claimed uremia as the cause, because he found albumen in the urine of sick cows, also renal elements, and on histological examination changes pointing to nephritis.

Schmidt (of Kolding), on purely theoretical grounds, put forward a new intoxication theory, according to which the origin of the formation of the poison was in the udder. Originally he believed that the poisonous material developed through the decomposition of the colostrum corpuscles which he believed to be epithelial cells; later he assumed toxins developing in the udder in some unknown manner, which after absorption into the blood produced a poisoning of the whole system.—According to Sonnenberg the milk contains substances which have a toxic effect on rabbits and the amount of which is increased in parturition paresis.—Hemprich imputed an intoxication to certain combinations of proteins that are not used up in the milk secretion, whilst according to Meyer, and also to Leclainche, the causes of milk fever might be sought in certain poisons formed in the body and only incompletely excreted through the udder. Finally Delmer accuses the absorption of poisonous material from the udder, which is formed there by the disintegration of protein substances, stimulated by some proteolytic ferment. In favor of this hypothesis the cases of illness similar to parturient paresis, but which develop independently of parturition may be cited.

The hypothesis advanced by Schmidt, as well as those others that were similar to it, were based solely upon the results secured by the treatment of the disease. As will be shown further on, however, the favorable results from these therapeutical methods are not at all adapted to prove the correctness of the above mentioned hypotheses. Authors other than Mamadysecky have not found any support for the uremic theory, and besides the degree of albuminuria that is occasionally present bears no relation to the severity of the symptoms.

If after all it appears to be most probable that milk fever arises from an intoxication it must, for the present, be left undecided whether the origin of the intoxication is to be looked for in the uterus or in the udder or in the entire organism. The experiences made concerning anaphylaxis admit the possibility that parturition paresis is merely a form of anaphylaxis

which has been brought about by the specific protein of the placenta or possibly of the glandular cells of the udder, and that the absorption of considerable amounts of placenta or milk protein produces anaphylactic symptoms. After a brief excitation of the cortex and of the sub-cortical centers, among them principally of the motor centers, the assumed poison produces paralytic disturbances of the function of the brain, and also a brain anemia without leading to tissue changes in the nervous system.

**Anatomical Changes.** Autopsy gives no characteristic findings. The mucous membrane of the vagina exhibits the punctiform hemorrhages seen usually after parturition, and is moderately edematous. The external os uteri is swollen and infiltrated with serum, studded with hemorrhages from a pea to a hazelnut in size, and more or less open. The incomplete involution of the uterus which was first pointed out by Hess is at times easily recognizable; the uterus contains some hundreds of grammes of a chocolate brown, reddish or yellowish odorless fluid. The internal os and the uterus proper are usually in a similar condition to that of the orificium uteri externum. The vessels of the abdominal organs appear more filled with blood than those of the other parts of the body. The central nervous system shows no changes with the possible exception of anemia. Secondary lesions may be sometimes noticed, such as muscle tears, portions of food in the air passages, foreign body pneumonia and exceptionally fractured bones (in one of Wilhelm's cases fracture of the second cervical vertebra).

**Symptoms.** In the great majority of cases the cows remain quite healthy from one-half to two days after a normal and easy parturition, and the activity of the udder proceeds regularly. Generally on the second, often on the third day or towards the end of the first day, seldom earlier or later, the appetite declines without any visible cause, and at the same time the animal exhibits a peculiar restlessness, looking about nervously, emitting a dull bellow and tripping restlessly with its hind feet. Exceptionally one notices also a more decided excitement, such as frequent bellowing, climbing into the crib, and at the same time spasms in single muscles, viz., in muscles of face and neck and also in the muscles of mastication, which, however, only continue for a short time, at most a few hours (according to Hess such cases belong to puerperal septicemia). Some degree of weakness soon becomes noticeable; the animal begins to stagger, spreads out the forefeet, trembles markedly, can finally not continue any longer on its legs and falls to the ground. The animal makes some attempts to rise but then a comatose condition develops and it lies still. At this stage the animal lies, usually half way over on her side, with feet drawn



under the body, whilst the head is turned sideways and rests on the shoulder of the upper side; if it is straightened and then let go it immediately falls back helplessly into its previous position. At times the head is stretched out with the under jaw propped on the ground, or the animal lies on its side with head extended and legs outstretched. All muscles seem to be relaxed, but not paralyzed, since the animal at times turns herself over or gets up on her knees and creeps forward.

The vital functions appear to be lowered in the animal, which lies on the ground as if paralyzed. The patient seems to have fallen into a deep sleep, is quite oblivious to her surroundings, so that she takes no notice of flies nor reacts to pricks or other irritation. The comatose condition results also in a decline or total absence of the reflexes as a result. Skin and tendon reflexes cannot be produced; only by tapping the muscles can twitchings be caused, because their mechanical irritability remains unchanged. Owing to the absence of the corneal reflex the periodic shutting of the eye orifice, which in consequence of loss of consciousness is half closed does not ensue, the cornea is dull in consequence, dry, and sometimes wrinkled, but at times the eyes are completely closed so that the animal appears asleep. The pupils are greatly dilated and fixed. The mouth is half open, the tongue protrudes, and its upper surface becomes dry. The absence of the swallowing reflexes produces a copious flow of saliva, and if medicine be poured in or if the contents of the rumen be regurgitated into the pharynx choking may occur, and easily lead to gangrene of the air passages, because the reflex of the larynx (coughing) is also extinguished.

The respirations become deep and slow, not seldom accompanied by groans, and later stertorous through vibration of the relaxed soft palate, or rattling in cases where edema of the lungs has already occurred, in both cases more or less labored. The activity of the heart is always hastened, 70 to 90 per minute, in severer cases up to 120, seldom arrhythmic, the pulse is small, soft, and occasionally only perceptible at the abdominal aorta (Hess).

There arises an arrestment, or at least a retardation, of evacuations, and consequently feces collect in the rectum and urine in the bladder. The contractions of the muscular tissue of the stomachs and intestines are sluggish or are, in most cases, entirely suspended, consequently tympanitis sets in after a short time, which is often severe, whereupon the animal belches and expels gases as well as at times thin fluid contents of the rumen into the cavity of the mouth. The urine often contains some albumin and sugar, the amount of which may be as much as 4% (Nocard). The investigations of Porcher show, however, that the sugar contents of the urine are of no special significance, because the urine of healthy cows and of other female animals often contains sugar after parturition, in the



form of lactose; less often grape sugar may be found as well as milk sugar.

According to the records of many observers the udder is flaccid, and always secretes little or sometimes no milk at all. Vaginal discharge is always absent, but the external os remains more or less open, and the already mentioned incomplete involution of the uterus can be demonstrated (Hess).

At first a moderate rise of temperature occurs at times, later, however, with the onset of the paralytic condition, the temperature always falls and may decline to  $35.4^{\circ}$  C. (Himmelstoss). Nelke attributes the low temperature to failure of action of the sphincter ani muscle; when he put the thermometer sufficiently far enough into the rectum he always found a temperature of between  $38.8$  and  $39.0^{\circ}$  C. The ears, horns and feet always feel very cold.

Zehl recognizes three forms of milk fever. In the subacute form digestive disturbances and dullness are prominent, whilst paralytic symptoms are absent or at most trivial (see also chronic intestinal catarrh of cows after calving, page 265). The mild form manifests itself by the exclusive presence of paralytic symptoms and corresponds clinically with sacral paralysis after birth (see page 748). In the third form both previous forms unite to bring about the true milk fever. How far the first two forms can be ascribed to milk fever later observations must determine.

**Course.** The form of illness described above usually develops within a very short time, even as early as 1 or 2 hours, and remains unchanged for a certain period, generally about a day. In favorable cases improvement usually occurs just as rapidly as the symptoms of illness have set in. The patient opens her eyes, looks about with uplifted head, moves her tongue, and makes swallowing movements, she passes dung and urine, her temperature returns to normal, finally she gets up and takes food. Recovery can occur without any treatment within a quarter to half an hour, often even in very severe cases where death was to be feared every moment (thus Hess saw some cows that appeared to be lost, recover in a few minutes after being turned over). In other cases the ailing animal exhibits some weakness for 1 or 2 days, or it may even be for 3 days.

In unfavorable cases death may occur almost unnoticed, the quiet breathing becoming gradually slower and finally being arrested along with the heart action. In other cases death is immediately preceded by symptoms of restlessness, perhaps by convulsions, or by a sudden copious diarrhea. Occasionally it happens that the animal after a short period of unrest suddenly collapses, lies as if lifeless and passes away under convulsions. In such cases a fracture of the bones of the skull or the vertebræ may be thought of (in a case of Wilhelmi's the animal struck its head on a piece of wood which had been placed under it and broke the second cervical vertebra).

In a number of cases the animal recovers after the disappearance of the paralytic symptoms, but one or two days later



signs of aspiration pneumonia become manifest, such as gradually increasing difficulty in breathing and a febrile rise in temperature, and these are followed by death in 4 or 5 days. The inflammation of the lungs can occur in the paralytic stage, so that one should always remember this if the breathing becomes more frequent and labored or if the internal temperature rises.

In exceptional cases, or at least in less severe cases, sequelæ of the illness remain (Jensen), such as paralysis of the n. ischiadicus or of the n. cruralis on one or both sides, further very rarely paralysis of the n. facialis. These paralyzes are traumatic in origin and disappear at times after a few days, or on the contrary after weeks or months, a more or less visible atrophy of muscle having occurred in the meantime. Exceptionally necrosis of the m. biceps femoris, of the m. semi-tendinosus or of m. gastrocnemius occur, and very rarely of the teats or the digits of the hind feet. After the use of the original Schmidt treatment (injection of pot. iodide into the udder), mammitis occurred in 2 to 4% of the cases, mostly with permanent drying up of the milk. A diminution in the milk secretion, lasting until the commencement of the next period of lactation and amounting to 2 or 3 liters daily, persists also after the injection of air into the udder, without the development of an udder disease.

**Diagnosis.** Loss of consciousness following immediately after birth and not accompanied by fever and the paralytic condition are so characteristic that the ailment can hardly be mistaken for another disease. Lumbar paralysis after parturition may be mistaken for it, but here the paralysis is limited to the hind quarters and consciousness is not lost (see page 748). Puerperal septicemia is characterized by local inflammatory changes in the genital organs, by a rise of temperature and by the symptoms of a febrile disease. The so-called railroad sickness (see page 790) may easily be distinguished from parturient paresis on account of the mode of its occurrence. Fatal hemorrhages in consequence of rupture of the uterus manifest themselves by the appearances of severe bleeding (see vol. I). Acute brain diseases (cerebral abscess, tuberculosis) might likewise occasion mistakes in diagnosis (Salvisberg).

**Prognosis.** The losses caused by milk fever varied greatly before the employment of the udder treatment, and this was partly because in some cases slaughtered animals were reckoned in the total losses; an emergency slaughter which renders a possible recovery impossible is undertaken more readily in one place, less so in another. On an average the fatalities ranged from 40 to 50%, but often much less or more. It is to the unquestionable and permanent credit of Schmidt that after the introduction of his method of treatment the mortality has in

general become much lower and now amounts to 15 to 25%, or after using air infusion exclusively, even to less than this (maximum 16% [Hess]). In a given case, however, it may be very difficult to foretell what the probable result of the illness will be, because on the one hand an apparently slight attack may end in death, whilst on the other hand an exceedingly ill animal often recovers. In general it may be said that the occurrence of the disease before, during or shortly after parturition as well as a very rapid aggravation of the condition must be considered as signs of an unfavorable prognosis (de Bruin, Hess). The presence of concurrent tuberculosis in a sick animal is also unfavorable.

A relapse may occur in the course of the next few hours or in one or two days, especially if after the udder treatment the air is removed too soon from the vessel or escape from it spontaneously.

**Treatment.** First of all the animal should, if possible, be provided with an airy place, well bedded and placed lying on her left side with head higher than the body. In order to prevent tympanites it is advisable to support the animal with straw bundles so that she lies slightly inclined to the left side with her feet under her breast bone. Constant watching is required so that she does not injure herself when tossing about.

The administration of medicine or other things by the mouth should be avoided entirely in order to prevent aspiration. If tympanites is severe the gases may be removed with the stomach tube or with a trocar. The accumulated urine may be induced to flow by slight pressure on the bladder.

Subcutaneous injections of vasoconstricting remedies may be very usefully employed. In the first place is caffeine (4-8 gm.), then strychnine (0.02-0.08 gm.), veratrin (0.1-0.2 gm. in dilute alcohol), eserine (0.10-0.15 gm.), or pilocarpine (0.2-0.3 gm.) give good results; eserine and pilocarpine cause also vigorous contractions of the intestinal muscles. This method of treatment when practiced by itself in early times, as well as also now when used in conjunction with Schmidt's treatment gives favorable results. Barium chloride can be usefully employed (animal experiments of De Bary, Marek); it exercises a powerful contractile effect upon the vessels, and may be used in small doses intravenously (0.05-0.10 gm. in 10 gm. of warm water) or as clysters (1-2 gm. in a liter of lukewarm water). Cutaneous stimulants like friction of the skin, fomentations with cold or warm water, etc., exercise a stimulating effect on the vasomotor nerves and tone up the whole nervous system.

The injection of air into the udder has become quite a general method of treating milk fever. At first Schmidt (Kolding), the founder of this therapeutic procedure, used 1% pot. iodide solution along with the admission of air, but nowadays this method of treatment has been wholly replaced by the more



convenient and cheaper inflation of the udder with air, which promises far better results.

**Technique of the insufflation of air into the udder:** The simplest form of apparatus consists of a hard rubber bulb connected with a hollow metal cylinder containing cotton wool, and an udder catheter or milking tube fastened on the end of a rubber tube (in case of necessity a feather quill); it is sterilized by boiling in water and rinsing out with a one per cent creolin or lysol solution. Zehl's double air catheter (Fig. 116) appears especially useful. The udder is prepared for the insufflation by milking out and thorough cleansing all four teats and the surrounding parts with luke-warm water and a soft brush. A clean piece of linen soaked in a 2% creolin or lysol solution is then placed under the udder and the teats and the neighboring parts are rinsed with creolin or lysol solution. Then the catheter or milking

tube is introduced into the canal of the teat and the bulbs are brought into play so that purified air is passed through the cotton wool into the udder. Sufficient air must be introduced so as to cause a plainly visible, tense, elastic stretching of the udder. In order to prevent the catheter from falling out, or the infused air from returning, the points of the teats must be compressed with the disinfected fingers; the rubber rings frequently employed appear to be less adapted for the purpose (Hess). At first the lower quarters of the udder should be inflated and afterwards the upper ones.

Knüsel uses 6-10 liters, and Salvisberg 12-15 liters of oxygen instead of air. The oxygen is held in a container, and after insertion of a regulating apparatus is allowed to pass into the udder and the proceeding is repeated if necessary; but this method of treatment has no advantage over that with atmospheric air (Hess).—Formerly some authors used salt or lysol solution instead of pot. iodide solution or ordinary water boiled and allowed to cool to body temperature, up to 4 liters (Peters &



Fig. 116. Double air-catheter, after Zehl, for the introduction of air into the udder.

Wessel have each injected intravenously 2 liters of a 0.5% pot. iodide solution warmed to 38° C, while others introduced this solution into the womb or gave it internally).

Since Schmidt's treatment has been employed the losses in parturition paralysis have declined from 50 to 25 and even to 10% and often lower. This applies not only to the original Schmidt treatment, but to its various modifications without reference as to whether the pot. iodide is introduced in the food or through other organs, or whether, instead of pot. iodide, gaseous substances or other fluids are introduced into the udder.



The Schmidt procedure—including its modifications—has proved a trustworthy causal and strikingly effective treatment of milk fever. Many weighty reasons may, however, be adduced to the effect that the supposed cause of the disease is not removed by this method. Above all it must be considered that some authors (Möbius, Meyer and others) have recorded considerable losses (up to 60%) in spite of the employment of this treatment. But the fact that the most varied materials introduced into the udder—potassium iodide, disinfecting solutions, neutral fluids and gaseous substances—in general all give the same favorable results, and that further potassium iodide when introduced by other channels into the system, likewise has a favorable effect, makes one have justifiable doubts whether all these materials were actually suitable to check the formation of the harmful toxins and to neutralize that portion of them that has already become absorbed. It must be recollected, also, that a part of the sick animals recover in so quick a time after treatment (after a few hours, in some cases after a few minutes), that the period would be too short for the inhibition of the presumable toxin formation and the neutralizing or elimination of the absorbed toxin.

If it is further considered that the method of treatment which, depending upon the inflation of air, permits a greater tension of the udder tissue, has yielded a better percentage of cures, it becomes more reasonable to assume that the inflation of the udder (which is very sensitive shortly after parturition) acts solely as an intensely irritating agent. In this manner it would on the other hand contribute to relieving the unconsciousness, and on the other hand stimulate the vasomotor nerves which in their turn are capable of producing a more decided emptying of the abdominal vessels or of raising the blood pressure. In contrast to this view many authors assume a mechanical action, namely, thus that the blood is believed to be pressed out of the udder, the tissue of which is put on a tension by the injection or the insufflation of air, and that the further flow of blood into the organ is interfered with, which then is supposed to lead to a removal of the supposed cerebral anemia.

As long as the nature of the disease remains obscure, it is self-evident that the manner in which Schmidt's treatment produces its effect cannot be definitely stated.

In a consideration of the curative results it must be remembered that, since the introduction of Schmidt's method in which the administration of medicines by the mouth is obviated, complicating inflammations of the lungs have become less frequent, and that, encouraged by the favorable results, owners do not slaughter the animals as readily as formerly. Many older records testify that similar and, indeed, more favorable curative results were observed before the application of the Schmidt treatment. Thus Krüy records 20% of losses, Porter about 19%, Black about 17%, Ajolfi about 10%, Breckbohm about 5.55% (among 72 cases 4 deaths), Harms about 2.7% and Biot in 200 cases about 2% of losses.

**Prophylaxis.** Considering the predisposing factors in the disease it seems advisable to give cows daily exercise during the period of pregnancy. Towards the end of gestation, especially if they are dry for a long time before calving and 6 to 8 days after calving, they should be fed more sparingly. De Bruin suggests that the cow be allowed to calve in the open in summer.

Albrecht noticed the disappearance of this oft recurring and serious disease on an estate, when the above mentioned prophylactic procedure had been carried out.

**Literature.** Albrecht, W. f. Tk., 1904, 48.—Bredo, Bull., 1909, 228.—Delmer, Bull., 1906, 417.—Gebauer, B. t. W., 1906, 377.—Habicht, B. t. W., 1907, 502.—Harms, Mag., 1891, 388.—Hess, Schw. A., 1905, XLVII, 279.—Hooper, Ann., 1905, 510.—Jensen, Z. f. Tm., 1898, III, 1.—Keim, Zur Atiol. d. Gebärpärese, Diss., Leipzig, 1909 (Lit.).—Mathé, Rec., 1884, 658.—Meier, B. t. W., 1907, 103.—Nelke, Über d. Kalbfieber d. Rindes usw., Diss. Bern, 1909 (Lit.).—Porcher, Rev. gén., 1903, II, 479.—Reinhardt, B. t. W., 1907, 150.—Salvisberg, Schw. A., 1908, L, 230.—Schmidt, W. f. Tk., 1905, 21.—Schmidt, Monh., 1898, IX, 241; B. t. W., 1902, 497.—Sonnenberg, B. t. W., 1907, 283.—Stahn, Z. f. Vk., 1906, 214.—Zehl, Die Gebärpärese d. Rindes, 1905 (complete Lit.); B. t. W., 1908, 117.

**Diseases Similar to Milk-fever.** Diseases occur occasionally, independently of parturition, although clinically they are more or less like



milk fever. The syndrome sometimes stands in causal relation to mammitis, (Lungwitz, Otto, Zehl) or nervous symptoms similar to those occurring in milk fever arise exceptionally as a result of overloading of the rumen, atony of the first 3 stomachs, or necrosis of the liver, and these appearances may affect bulls or bullocks as well as cows (see pages 253; 269; 529). More frequently, however, a primary onset of the affection is noted in good milking animals. In such cases it has frequently been noted that a one-sided feeding, with much concentrated material like cottonseed-meal, brewers' grains, clover, hay, etc., is capable of giving rise to a clinical picture similar to parturient paresis (Hansen, Rasmussen, Seiderer, Meier, Gratia). Whether the last mentioned causes form the foundation of the primary cases hitherto noticed or whether other causes play a rôle remains undecided.—To all appearances the affection may be considered as due to an intoxication.

The treatment is similar to that pursued in parturition paralysis and has as a rule about the same result.

**Literature.** Gratia, Ann., 1904, 387.—Giniésis, Bull., 1905, 62.—Hansen, Maanedsskr., 1905, XVII, 177.—Lungwitz, S. B., 1895, 97.—Meier B. t. W., 1904, 89.—Otto, S. B., 1899, 87.—Rasmussen, Maanedsskr., 1905, XVII, 270.—Seiderer, W. f. Tk., 1905, 21.—Zehl, B. t. W., 1906, 297.

**Railroad Sickness of Cows.** (So-called emptiness of the rumen, railroad fever.) By this name a peculiar diseased condition, similar to that seen in milk fever, is designated, which occurs often in cows that are far advanced in calf, and also in well fattened animals during or after a long continued transport by rail. Voigtländer first recorded it, and later it has been observed by Weigel, Röder, König, Noack, Estor, Villagio, Otto and Steffani. Recently Schmidt has been occupied with a careful study of the disease.

**Etiology.** Of the causes of the illness it is only established that it is connected with long sojourn and standing in crowded and hot railway cars without food and water. Thus Estor and Schmidt noted the illness almost exclusively during the period from April to September, or after a railway journey lasting over 24 hours. Further, pregnancy is of special importance and according to Schmidt a railway journey undertaken by cattle immediately after being brought up from pasturing has an effect in producing the illness, and he has observed it only occasionally where cows have been previously stabled.

Voigtländer believes that an empty rumen is the cause, whilst Estor assumes also an exhaustion with the accumulation in the system of the products of fatigue resulting in autointoxication. According to Villagio the disease is primarily a muscular affection with secondary nerve trouble.

Schmidt declares the **pathogenesis** of the illness to be as follows: As a consequence of the increased muscular activity during railway transport, there results an increased flow of blood to the muscular tissue of the extremities and of the body, in advanced pregnant animals, also to the uterus and udder, in consequence the amount of blood in the other organs diminishes. Anemia then develops in these organs and is increased by the fact that the blood vessels of the skin dilate and the amount of blood in the body diminishes considerably, because on the one hand no water is taken up and on the other hand an increased output of water occurs. If this condition lasts more than



24 hours paralysis of the vasomotors sets in, and high temperature results in the neighborhood from the dilation of the vessels and the increased muscular action.

The **autopsy** reveals no characteristic lesions. The rumen contains very little food or is empty (Voigtländer). The muscles of the loins may be edematously infiltrated (Estor) or they, as well as the muscular tissue, of the body in general may be colored brownish red or brownish black, whilst fatty infiltration of the parenchymatous organs may be noted (Noack). The chemical reaction of the muscles of slaughtered animals is, according to Estor, always alkaline.

The **symptoms** of the illness usually arise at the end of a transport, but often earlier, that is during the journey. The animals exhibit considerable unrest in look and movement, the gait being stumbling and staggering and the tail extended. Shortly after this the hind parts collapse, although for a short time the animal makes efforts to stand up. Soon it remains lying down with its hind extremities drawn up, but frequently changing its position. The mucous membranes appear considerably injected, the pulse is quickened, its number goes up to 120 beats a minute. The body temperature varies between 38.8 and 39.4° C. The breathing is always quickened (up to 40 respirations per minute) labored, and later stertorous. Peristalsis and appetite are in abeyance, whilst the feeling of thirst may sometimes be increased; voidance of dung is lessened or completely at a standstill. Frequently violent labor pains occur which are not depending on parturition. Estor found in many cases an amount of 1 per thousand of albumin in the urine. The udder is filled but soft and contains a considerable quantity of normal milk. Consciousness seems at first undisturbed, but later it becomes upset more and more; the animal turns its head on its side, closes its eyes, becomes indifferent to all irritation, and in this condition presents a picture like that of milk fever. Ninety per cent of the cases end unfavorably in 1 to 2 days with aggravation of the paralytic symptoms and dyspnea so that the animals usually have to be slaughtered or die. With suitable treatment (Schmidt) the patients recover almost without exception.

The **treatment** is similar to that in milk fever. Schmidt has obtained cures in almost all cases by the insufflation of air into the udder.

**Literature.** Estor, D. t. W., 1899, 233.—Hartenstein, S. B., 1893, 126.—Röder, *ibid.*, 1892, 101.—Schmidt, B. t. W., 1906, 775 (Lit.).—Voigtländer, S. B., 1878, 88.

## 6. Catalepsy. Catalepsia.

By catalepsy a peculiar state of illness is meant in which the animal is incapable of performing voluntary movements for a certain time, because in consequence of stiffening of the muscles, certain limbs remain for a long time in a given position, which may, however, be changed passively at will. This symptom, which is rare in man, and which when occurring is often a symptom of hysteria or a result of certain psychoses and organic diseases of the brain, has been noticed in isolated cases in animals. It appears from the records, however, that the symptom did not originate as a distinct neurosis, but merely as the resulting condition of organic diseases of the nervous system (compression of the spinal marrow, inflammation of the coverings of the cord, diseases of the brain), or of severe intestinal disorders. In consequence one is not justified in ascribing these cases to a catalepsy in the strict sense of the

word, but it appears suitable to designate them as "cataleptic stiffening of the limbs."

The attack occurs either only once or it is repeated, the animal suddenly stiffening in the position and attitude which it assumed immediately previously in walking or lying, and thereafter is unable to execute any further movement. The muscles feel tense and firm, their outlines are sharply defined, there is increased resistance to passive movement, but the extremities, head or neck may be put in another position without any great effort, and they will remain in these positions even if the equilibrium of the body is thereby endangered; one receives the impression of the moving of a plastic flexible mass (waxy flexibility, *flexibilitas cerea*).

Treatment depends upon the cause of the illness.

**Literature.** Fröhner, D. Z. f. Tm., 1883, IX, 119.

## 7. St. Vitus's Dance. Chorea.

(*Sydenham's Chorea; Chorea minor. s. Scti Viti.*)

As St. Vitus's dance is designated a neurosis which is mostly transitory and occurs chiefly in childhood, being characterized by involuntary and arrhythmic but irregularly distributed twitchings of muscles of manifold combinations, frequently connected with psychic disturbances. The abnormal movements are in themselves coordinated combination movements (grasping, defensive, or movements of expression), yet at the moment of their occurrence they are always without purpose, and sufficient to disturb other intended movements. During sleep the convulsions usually disappear entirely.

A more or less similar clinical picture is said also to occur in animals, yet in most cases organic diseases of the nervous system or of other organs of the body must be assumed as fundamental affections, and therefore they must not be classed with St. Vitus's dance proper. Thus in the course of distemper or after the cessation of the acute symptoms of the disease, which sometimes are not noticed at all, local spasms may occur, which by French authors and recently also by Joest have been designated chorea, but not justly so, and still less as a distinct disease as Joest has proposed. Clinically there is a great difference between chorea and the spasms in distemper. In distemper the twitchings are continued in the same group of muscles or limited to the region of certain nerves in which they originate; they exhibit a more or less decided rhythmic character and are continued and lasting. The peculiarity of distemper spasms has been shown by repeated microscopic examinations to be due to disseminated myelitis or encephalitis, that is an organic disease of the nerves. Dexler had already explained spasms of distemper in this manner, and recently this author has shown by myographic sketches that the muscle convulsions are similar to those seen in the so-called "tic," which disappear with consciousness, and are influenced by conditions which cause psychic disturbance.

Sometimes spasms are due to severe intestinal catarrh or inflammation, and occasionally to other diseases (in the case of Liénaux an abscess in the neck) which spasms, however, cannot either be classed with

chorea. In a great many of the remaining recorded cases in cattle, exceptionally also in horses and swine, the character of the twitchings was not that of chorea and they will therefore not be considered further.

Only in a very small number of the cases formerly described as chorea were the twitchings similar to those of true chorea; still the nervous system was not examined properly, so that its organic disease or that of such another organ is, even in these cases, not completely eliminated as the basis of spasms similar to those of St. Vitus's dance. (Dexler, D. t. W., 1909, 314 [Lit.].—Joest, Z. f. Tm., 1904, VIII, 179 [Lit.].—Poenaru, Bull., 1908, 671.)

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**Chorea Electrica Congenita.** Besnoit observed a peculiar congenital neurosis in a lamb which descended from a flock affected with trembles.

Strong and quite rhythmical twitchings in the muscles of the head, neck, body and extremities occurred five or six times in a second. In consequence the head was moved like a pendulum or from side to side, at times there was grinding of the teeth, whilst the tail executed slight trembling movements. The extremities were abducted and then adducted, then flexed and again extended, as a result standing up was mostly impossible, and the animal appeared to be paralyzed. During the movements the twitchings increased in intensity. On passive pressure of the head to the chest wall the spasms disappeared. When lying half on one side the head performed scarcely perceptible quivering movements, whilst during sleep all spasms disappeared. External and psychic irritation caused a considerable aggravation of the twitchings. After 8 weeks the lamb recovered completely without having exhibited other signs of illness in the interval.

Besnoit identifies the affection with the chorea electrica (Bergeron) in man, which occurs preferably in childhood. The trembles could in his opinion have played only a predisposing rôle. (Besnoit, Rev. vét., 1906, 433).

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**Chorea in New-born Animals.** In young pigs peculiar twitchings limited to certain groups of muscles are not uncommon. These spasms occur immediately or shortly after birth, and at times appear to be of an hereditary nature. Thus Hess noticed the illness in the litters of three sows that had been served by the same boar, whilst Scheller saw all the young of one sow affected. On the other hand the rôle played by external influences in causing the complaint cannot be disregarded, for Kühn saw an enzootic spread of the disease in which the pigs were kept in cold pens and exposed to cold winds. Carbaret has described a similar form of disease in a new-born foal.

The symptoms consist in that the flexor muscles of the posterior or of all extremities perform quick, powerful twitchings, about 100 to 140 in a minute, and in consequence the animal executes peculiar dancing movements whereupon the head moves passively in conjunction with these, or becomes likewise affected with spasms.

In most cases recovery takes place at the age of 6 to 8 weeks.

**Literature.** Cabaret, Rec., 1905, 719.—Hess, Schw. A., 1884, XXVI, 240.—Kühn, Pr. M., 1855, 56, 113.—Scheller, *ibid.*, 1853-54, 101.



### 8. Spasm of the Nerves of the Diaphragm.

(*Spasm of the diaphragm, so-called abdominal pulsation; chorée du diaphragme* [French].)

Spasm of the nerves of the diaphragm consists in twitching of the muscles of the diaphragm.

**History.** The condition which produces convulsive jerking of the body was for a long time attributed to palpitation of the heart or to increased pulsation of the aorta, but recent observations have shown that it is due to muscular spasms which are even at this time very often mistaken for spasms of the abdominal muscles. With the aid of a considerable number of available observations Thomassen made a careful study of the illness, and more recently Zürn has undertaken an analysis of the symptoms in two cases.

**Occurrence.** Spasm of the diaphragm is not uncommon in horses, but is observed exceptionally also in cattle (Paimans, Bach, Lucet) and also in dogs (Marek). The greater number cases described under this name belong without doubt to spasm of the abdominal muscles.

**Etiology.** Rather often digestive disturbances, or acute stomach ailments precede spasm of the diaphragm or it occurs as an accompanying complaint to intestinal catarrh or stoppage of the bowels. In these cases the spasm is probably a reflex condition being produced by an irritation occurring in the mucous membrane of the stomach and intestine being communicated through the n. vagus to the medulla oblongata, and from there to the diaphragmatic nerves, and possibly also to the nerves of other muscles. The effect of chemical substances absorbed from the intestinal canal does not appear to be excluded, in fact, it appears probable in many cases.

In some cases the spasms are not dependent on digestive disturbances, but may arise after excessively severe work or after psychic excitement. In these cases Haubner & Siedamgrotzky believed that the diaphragmatic nerve was irritated by the agitation due to strong beating of the heart, or that for some reason the nerve had become more irritable. Thomassen and Langendorf on the contrary believe that the nerve which passes over the heart muscle is irritated by the electrical current generated at every systole, the irritation being capable of causing a spasm only in a nerve that had become irritable.

The connection between the heart movements and the spasms of the diaphragm is indicated by the fact that the number of the two contractions in many cases coincide, or in cases where the contractions of the diaphragm occur less frequently, these immediately follow upon the heart beats.

But even if such a connection exists, the spasm of the diaphragm cannot be attributed without further evidence to the heart action, as is shown by a case of the

authors in a dog. In this dog, which was suffering from catarrh of the stomach and bowels, spasms of the diaphragm occurred in equal number to the heart beats and immediately following these; besides spasms were observed, far less often and at quite irregular periods of time, in different muscles at the head, neck and limbs. The common origin of the spasms was plain enough here, and yet the contractions of the diaphragm followed the heart beats.

At times the spasms are observed in acute inflammatory diseases of the thoracic organs, especially of the serous membranes or of the pleuræ. Finally myelitis in the neighborhood of the center of the diaphragmatic nerves may occasion spasm of the diaphragm (personal observation).

**Symptoms.** The disease is manifested by peculiar rhythmic jerkings of the body, which are strongest along the arches of the ribs, and at first sight give one the impression of very strong heart beats. At each convulsive movement there is a depression in the neighborhood of the costal arch, whilst at the same time the portions of the abdominal wall, which are situated further back, especially in the region of the epigastrium the flank, bulges out somewhat; the intercostal spaces on the contrary, along the whole extent of the lower part of the chest or only in the posterior third of the chest show spasmodic retraction. With the flat hand one can distinguish short powerful beats in the region of the lower ribs, and these become gradually weaker in all directions.

The number of impacts may coincide with the heart beats and then the convulsive jerkings immediately follow upon the heart beats. With an accelerated heart action the movements become more frequent; far more often, however, the number of pulsations is higher or lower than the heart beats, or no regular relation between the two can be established. In two cases of the authors the spasms of the muscles of the back ceased for a short time on percussion of the back. From the nasal orifices one may distinguish a sound similar to that of a sobbing, but it must not be mistaken for that which is caused in clonic spasm of the abdominal muscles, by the jerky and short expirations. The animals appear restless and generally show no appetite. The condition hardly ever lasts longer than two days.

**Diagnosis.** The only characteristic signs of spasm of the diaphragm are the bulging out of the epigastrium and flank, which are synchronous with the convulsive jerkings, and at the same time the simultaneous retractions of the intercostal spaces. In doubtful cases the hand introduced into the rectum up to the place where the diaphragm is attached will immediately detect the convulsive movements of the latter. The clonic spasm of the abdominal muscles may be distinguished in that one can see the contractions of the abdominal muscles, or feel them with the hand placed in the flank, and moreover a retrac-

tion of the epigastrium and a bulging out of the intercostal spaces occurs synchronously with the convulsive movements.

**Treatment.** The increased irritability of the diaphragmatic nerves and the spasms themselves may disappear without any treatment. Only Perrin noticed a fatal result in the horse. As a rule it is sufficient to keep the animal quiet, yet one can hasten improvement by subcutaneous injections of morphine (0.30-0.50 gm. for horses, 0.01-0.02 gm. for dogs), because the spasms cease entirely in half to one hour after a single injection. Any digestive disturbances that are present must of course be remedied (change of food, internally neutral salts).

**Literature.** Dupas, Rev. vét., 1906, 548.—Klingberg, Z. f. Vk., 1906, 23.—Perrin, Bull. spéc. des vét., 1906, 49.—Thomassen, Ann., 1892, 17 (Lit.).—Zürn, D. t. W., 1905, 25.

## 9. Local Spasms in the Muscles of the Head, Body and Extremities.

(*Tic.*)

Clonic spasms or twitchings occurring in the muscular tissue of different parts of the body are characterized by the fact that they are never associated with disturbances of consciousness, they are limited to single muscles or to the territory supplied by single nerves, and that a certain uniformity and regularity may be observed in this case, or even when several parts of the body are affected. These peculiarities distinguish this form of spasm on the one hand from epilepsy and eclampsia, and on the other from chorea, for which it is frequently mistaken. Most cases recorded in literature under the name of chorea belong to this category.

**Etiology.** In certain cases which need here not be considered in detail, the cause of the spasms lies in organic disease of the central or perhaps exceptionally the peripheral nervous system. As neuroses the convulsions may be occasioned by poisons which are not known exactly. This must be considered when the spasms are due to intestinal inflammation, catarrh, digestive disturbances or to other diseases, as is the case not infrequently (in one of the authors' cases purulent pleurisy). In many cases, however, they are perhaps due to reflex causes. Thus Liénaux noticed extensive spasms after opening an abscess on the neck of a dog, but they ceased after packing of the abscess cavity. A similar occurrence was noted by Krammel after docking the tail of a horse. Gunning saw the occurrence of such spasms in a horse after castration (after separation of the spermatic cord recovery resulted), whilst in Bartolucci's case spasms limited to one-half of the body were occasioned by



a trauma. In a considerable number of cases the causes remained unknown (as for instance in cases described as chorea by Anacker, Schleg, Albrecht and Besnoit).

**Symptoms.** The spasms occur almost exclusively in the form of twitchings, with which only very rarely short tonic spasms are associated. The number of twitchings is very variable and may be equal or different in all the involved muscles. Rather often the twitchings are more or less plainly rhythmic; quite irregular contractions, now in this and then in that muscle, as in chorea, are never observed. The spasms usually continue during rest, but as a rule occur less often, and are less powerful, than when some external influence arouses the attention of the animal. They are usually transient and disappear after a few days or several weeks. With the exception of those spasms that arise from severe basic diseases they occasion little or no disturbance of the vital functions of the animal, and do not impair their working power unless they involve a considerable area.

The clinical picture varies according to what muscles are attacked, and how many are affected. Spasms in the region of the facial nerves will draw the lips back by jerks and close the eyelids, etc. Uni- or bilateral spasm of the muscles of mastication will cause gritting of the teeth, which may be audible some distance away (Dexler, personal observation). If the neck muscles are attacked the head nods or moves convulsively to one side, whilst spasm of the back muscles causes curvature of the back upwards, downwards, or to one side. Bilateral spasm of the oblique abdominal muscles causes contractions of the abdominal wall, depression of the epigastrium, and a visible bulging of the intercostal spaces, or also an upward curvature of the loin region; a unilateral spasm on the contrary produces a curvature of the body to the same side besides contractions of the abdominal wall on the affected side. If the extremities are included in the attack then the joints are bent at times or are extended by jerks, whereby peculiar dancing movements are produced so that the animal lies down frequently or constantly. Contractions of the muscles of the skin coincide with those seen in getting rid of flies. In given cases the spasms occur in manifold combinations. In one case in a horse the authors succeeded in relieving the spasms of the muscles of the back for a short time by tapping the back.

**Prognosis.** In spasms arising from organic disease of the nervous system or from deep seated inflammation of the digestive organs, the prognosis is unfavorable in accordance with the nature of the primary disease, but convulsions from other causes mostly disappear after a short time, at the latest after a few weeks.

**Treatment.** Above all one should endeavor to find out the cause of the complaint. Besides, irritability of the nervous system may be counteracted with narcotics (bromine, chloral hydrate, opium, morphine). The spasms generally disappear, however, in time without any treatment.

**Literature.** Kramell, Z. f. Vk., 1905, 498.—Liénaux, Ann., 1897, 479.—Pohl, Z. f. Vk., 1909, 225.—Villemin, J. vét., 1905, 601.—Zürn, D. t. W., 1905, 25.

**Tetany.** (Tetania, Tetanus intermittens.) As tetany one designates in human medicine, according to Strümpell, attacks of tonic and generally painful spasms which in most cases occur symmetrically on both sides, and which preferably affect the flexor muscles, whereas the body, neck or head muscles are seldom attacked. In the intervals between the spasms, the electric, and especially the galvanic, as well as also the mechanical irritability of the peripheral nerves is increased. A further important diagnostic sign is that the spasm can be brought on by pressure on the trunks of the great arteries and nerves. The outcome of the disease is usually favorable. This neurosis has hitherto been noticed chiefly in nursing women, further in connection with certain acute diseases, as well as frequently after the extirpation of goitre, and in dilatation of the stomach.

Gunning (Am. v. Rev., 1895, No. 12) claims to have noticed tetany in a recently castrated horse in which adhesion had occurred between the spermatic cord and the scrotal wound; the tetany disappeared immediately on separation of the adhesion. Since the horse was not examined for the characteristic symptoms of tetany the case cannot be accepted as proving the occurrence of tetany in animals.

**La Tembladera.** This is an intoxication disease occurring very frequently in herbivorous animals in the Argentine Republic. According to Rivas & Zanolli this disease is brought about by a thread fungus parasitic on *Festuca Hieronymi*, but indigenous animals are immune. Six to ten hours after eating the plant fibrillary spasms arise in different muscles, likewise dullness and roughness of the coat. After a few hours swaying movements of the whole body occur both laterally and lengthways. At first these convulsive movements are but slight, but after a day or more they become so strong and occur so suddenly that the animal can only preserve an upright position with great difficulty, and at times falls down. After a further 2 to 7 days the patient remains lying down but shows spasms or muscular stiffness in the neck and extremities. After a decided rise in the pulse rate the body temperature sinks as low as 34° C. and in 4 to 14 days the animal dies, unless appropriate treatment (employment of purgatives, pilocarpine, arecoline, eserine) is undertaken. (Rivas & Zanolli, La Tembladera, Revista de la Fac. de Agron. y Veter., 1909, V.)

#### 10. Basedow's Disease. Morbus Basedowii.

(*Goître ophthalmique* [French]; *Grave's disease*; *Exophthalmic Goître*.)

**History.** In veterinary literature there are few references to this disease since it was first observed in man by Basedow in the year 1840. Jewsejenko, Cadiot, Marek, Ries have noticed a similar kind of illness

in the horse. Röder and Görig each in a cow, and Jewsejenko and Sonnenberg each in a dog. Even if all these cases do not exactly correspond in character to Basedow's disease, yet a few, and especially the case reported by Albrecht, make it appear probable that the disease also occurs in animals.

**Etiology.** In recent times disease of the thyroids has been considered in human medicine to be a primary affection since in consequence of hyperplasia they secreted more abundantly (hyperthyrea) and perhaps also produced a toxin (dysthyrea) which toxic material, on circulating in the blood, caused vasomotor disturbances (Möbius). According to this view, therefore, the illness is an intoxication brought about through an abnormal function of the thyroid glands. This theory which has the most adherents is still further supported by the fact that loss of the thyroids causes an exactly opposite clinical picture (cachexia strumipriva).

**Symptoms.** The disease is characterized by three cardinal symptoms, namely, struma, exophthalmus and tachycardia. First of all the enlargement of the thyroids is said to make its appearance. In the cases hitherto observed, one or the other or both lobes of the thyroid gland were enlarged to twice their size and more, smooth, of firm consistence and tense; the enlarged lobe may reach to the middle line, and may extend well backwards (Albrecht found on dissection three supplementary glands which were united to the enlarged thyroids).

Through exophthalmus (protrusion of the eyeballs) the expression becomes peculiarly staring and anxious. This causes the orifice of the eye to be opened too widely, and consequently the lids close less frequently and incompletely. Besides, a flow of tears is noticed, and on raising the head the upper lid does not follow the movements of the eyeball (Albrecht). Röder and Görig have observed a marked strabismus convergens in cows which, however, indicates the presence of an organic brain disease.

Palpitation of the heart may generally be noticed from a distance, every heart beat shaking the left side of the chest or the whole body noticeably; sometimes one sees besides a pulsation of the superficial arteries (Cadiot).

These symptoms are not always present in like degree, the exophthalmus, and still more the palpitation of the heart disappearing altogether temporarily. The rapid and paroxysmal onset of the disease may be accompanied now and then by other symptoms, such as timidity, trembling and languor.

**Treatment.** Quiet, cold applications to allay the heart palpitation, internally digitalis or narcotic agents may be employed. In obstinate cases partial extirpation of the thyroid glands may be tried, which experience shows has repeatedly



given good results in man and is also without danger in animals as a case of Ries's proves.

**Literature.** Albrecht, W. f. Tk., 1895, 233.—Bircher, *Ergeb. d. Path.*, 1894, I, 1, Abt. 5.—Cadiot, *Bull.*, 1892, 138.—Görig, D. t. W., 1898, 306.—Marek, *Vet.*, 1894, 310.—Ries, *Rec.*, 1899, 145.—Röder, S. B., 1890, 77.—Sonnenberg, B. t. W., 1906, 554.

**Enzootic Cretinism in Animals.** In regions where endemic cretinism occurs in man (especially in the enclosed valleys of the Alps, Pyrenees, in Franconia and in the Palatinate) frequent cases of cretinism were also observed in dogs, and were investigated by Cerletti & Perusini, v. Wagner and by Dexler. The disease may possibly occur in the other domestic animals also, although the cases hitherto described under this name probably belong to chondrodystrophy (so-called fetal rickets). Von Hansemann saw a case of cretinism in a jackal.

The cause of cretinism is found in a congenital complete failure of the function of the thyroids in consequence of disturbances in development or disease of the thyroid gland under the influence of unknown local injurious factors. Disturbances of metabolism thus caused result on the one hand in anomalies in the growth of the bony system and the soft parts, and on the other hand in disturbances of the development of the brain, and psychotic symptoms due to them.

The symptoms consist usually in a moderate enlargement of the thyroid glands, by a short and compact spinal column, with a large and short skull; the extremities are short and clumsy and the neck is short and thick. Rolls of skin are seen, especially on the head and neck, which cause the anterior part of the body to seem developed excessively in comparison to the hind part. Such animals frequently suffer from digestive troubles. Psychic abnormalities are especially prominent, being chiefly characterized by apathy. Although the senses are not much impaired the voice of the animal is strangely weak and it is apathetic, its ability of observation and of association is impaired, and its whole demeanor stupid, sleepy and lazy (Dexler).

A condition similar to cretinism may be induced experimentally by thyreoidectomy (*cachexia strumipriva*), as the experiments of Lanz, and especially those of Zietzschmann prove. This is true particularly in young animals, whilst adult animals become ill only after many months, and present a somewhat different clinical picture.

By treatment with thyroid gland these symptoms can, as a rule, be made to disappear completely or in a great measure. The thyreoidinum depuratum (1 dessertspoonful of the solution 0.1:100 internally) or the thyreoidinum Poehl (0.3-0.6 gm. 3 to 4 times daily) may be used for this object.

**Literature.** Dexler, B. t. W., 1909, 391, (Lit.).—Zietzschmann, *Mitteil.*, a. d. *Grenzgeb. d. Med. u. Chir.*, 1908, 353 (Lit.).

## 11. Psychoses.

As true psychoses (mental disorders) are designated in human medicine diseases of the cerebral cortex which are diffuse and usually only functional. They develop independently, run a chronic and afebrile course and are characterized

exclusively or principally by abnormal symptoms of psychic life (Psyche).

There is no doubt that most organic diseases of the brain, produce secondary psychotic symptoms, if it so happens that the cerebral cortex is involved in sympathy with the psychic processes, yet these diseases cannot be reckoned as psychoses in an exact sense, because on the one hand their nature depends on the organic change referred to, and because on the other hand the psychotic symptoms only form a part of the expression of the disease and the bodily (somatic) symptoms remain in abeyance. If the last mentioned cases were considered as true psychoses then most diseases of the brain must rank as psychoses.

Some veterinary authors have endeavored to prove by certain symptoms that true psychoses can occur in animals. But the question remained unanswered how many of the visible actions of otherwise healthy animals depend on psychic processes or on the contrary on reflexes, automatisms and fatigue, which of course have nothing to do with the psychic functions. Finally a new analysis of the observed symptoms was undertaken. Dexler has shown that only the occurrence of organic diseases of the brain can be considered as proved, and that these diseases may be connected with more or less pronounced psychotic symptoms, which, however, may not be mistaken for independent psychoses. The study of the symptoms arising in given cases must be approached circumspectly with proper consideration of the direction advanced by Dexler and of the elements of comparative psychology and general psychopathology, and one must hold strictly to the accepted definitions of psychiatry if one wishes to avoid confusion in the matter. It is further necessary to keep in mind that the nervous system of animals is not exposed to nearly so many injurious influences as is that of man in whom the various means of enjoyment, also certain infectious diseases (especially syphilis), affect the nervous system frequently and for prolonged periods on account of the comparatively long life. The reaction of civilization upon the psyche becomes manifest in man in an increasingly greater degree and no natural selection opposes the extension of morbid tendencies by heredity, as is usually the case in animals when certain transmissible anomalies make their appearance. For these reasons true psychoses can be expected in animals only very rarely if they occur at all, and their diagnosis requires above all the elimination of organic brain diseases by means of careful clinical and, especially, histological examinations.

The psychotic disturbances which hitherto have been observed in animals have been taken to correspond with the following psychoses of man.

To **Traumatic Early Psychosis** (a traumatic dementia) the cases described by Pierquin (1838; cited by Cadot) as "accidental dementia" are said to belong. Thus a young cat after a fall into a well suffered all through its life from weak mental faculties. A talkative parrot crept into a hiding place during the long continued thunder of cannons, and when later he was taken out of his hiding place he had lost his "vocabulary," and during his whole life afterwards could only make an attempt to imitate the noise of the cannon. In the first case there was probably a



chronic disseminated distemper encephalitis, and in the parrot a traumatic lesion of the brain. v. Kalischer has proved experimentally that injury of a definite part of the mesostriatum in parrots abolishes their power of speech.

In a case observed by Pierquin a previously healthy and very lively young cat was attacked with something like an "anxiety psychosis"; the animal became as if fascinated at the first sight of a dog; it watched the dog with anxious look, was motionless and stupid, and recovered only after several hours, the dog having been removed in the meantime. In this case there appears to have been simply a violent fright of a more than usually sensitive animal, similar to that in the so-called "fainting goats" (see page 775).

In this category would also belong the mad rush of a herd of animals (taking fright in a body, stampede, animal panic). But there is no valid reason for considering these as acute psychoses, because in such stampedes it is less an unthinking factor, the sensing of a danger and the transmission of this idea upon all individuals of a herd, that is here active, but rather reactions to external influences, based upon instinct (Dexler). Stampedes, especially in horses, and not seldom in cattle, camels and mules are not rare moreover, and may be very fatal in the course of war or manoeuvres. The impetus to a stampede is apparently always afforded by the abnormal excitement of one or several individuals and finally it causes an unreasoning flight in which the animals run blindly against obstacles or precipitate themselves into fire or water, etc. A blind, unreasoning flight is now and again noticed in single animals.

Straaten saw paroxysmal attacks of nervous symptoms in 12 cows of different herds after a severe fright. These were manifested by shaking of the head, staggering, falling down, stretching of the limbs, loud bellowing, labored breathing and diarrhea. In half an hour the attack was over.

To the affective psychoses especially melancholy, a case is supposed to belong which was observed also by Pierquin in a dog. An old dog went about sorrowfully after the death of his master, took insufficient food and finally became affected with marasmus, dying in a few months. Neither during life nor after death were the various organs examined for any derangement, and therefore internal illness leading to nutritional disturbances was not eliminated.

The three cases of psychic paralysis in dogs recorded by Arueh (1889) exhibit a great similarity to disturbances caused by compression of the spinal cord. Nor can exact proof be deduced from the case of supposed psychic paralysis in an epileptic horse, recorded by Girotti, since no anatomical examination was made.

Albrecht (1903) reports upon nervous disturbances in a dog whose extensor muscles of the fore and hind limbs together with the muscles of mastication refused to act after certain influences. By strong stroking or tapping of the back, the disturbances were made to disappear. When these attacks became more and more frequent, the dog finally became incapable of standing upright, and fell away rapidly in condition. As the examination of the brain gave negative results (the spinal cord was not examined) a psychic illness with pronounced symptoms of inhibition was supposed to have been present.

Staggers of horses was in many instances mistaken for a kind of "circular delirium." Gleisberg (1865) says in his text book of Comparative Pathology, that "idiopathic delirium" among animals, especially horses, is represented by staggers; he identified the so-called mad staggers with the paroxysms of "idiopathic delirium." On the other hand, Vogel (1888) grouped the maniacal symptoms together under the name of "mania transitoria" and considered them to be due to organic disease of the brain, nervous predisposition, sexual excitement and so forth. Zürn (1899) agrees with Gleisberg's view concerning staggers in horses, and Hoffmann (1899) also classes staggers and restiveness with the mental diseases without, however, attempting an analysis of the symptoms. Finally, even a prominent psychiatrist like Féré (1895) considers that staggers of horses is very similar to the "mental confusion" of man. But if one considers that the circular delirium of man in the maniacal stage is characterized by a hilarious ill-temper and by acceleration of cortical association, and in the melancholy stage, by morbid primary depression, primary inhibition of thought and frequently also by motor inhibition (Ziehen), the idea of allying staggers with the psychoses in man that have been mentioned, will have to be given up, the more so, as staggers is based upon organic brain disease.

By many authors the disseminated subacute or chronic encephalitis of distemper has been taken to be *dementia paralytica* (paralysis progressiva). In a case described by Cadiot (1896) in a three year old dog, previously very lively, which had been forgotten in a railway carriage and in consequence made a journey of 79 hours' duration, a certain idiocy developed after a few weeks, while a two year old dachshund observed by Nissl, suddenly became ill with nervous symptoms. Both cases call to mind the appearances of distemper encephalitis, and the histological changes



found by Nissl coincide with those found later by Dexler after a painstaking analysis. The same applies to the cases of sub-acute meningo-encephalitis of Marchand, Petit & Pécard, in which the authors draw a parallel between the histological changes found and those encountered in paralysis progressiva in man. But in reality the clinical as well as the anatomical changes in dementia paralytica are fundamentally different from those of the above named forms of encephalitis.

Further **hysteria** is also said to occur in animals. This was first asserted by Higier (1898) in connection with his observations in a cat and a canary bird. In both animals a paralysis arising from traumatic causes disappeared after a severe fright. The assertion is not supported by detailed clinical evidence, and an histological examination which might perhaps have shown evidences of some organic disease of the nervous system (traumatic lesion) was wanting. In a canary bird Losonezi noticed a loud chirping and then a lifeless appearance lasting for a short time, whenever the cage of the bird was taken down; the first attack occurred, as in Higier's case, after a cat jumped at the cage. Grobon likewise claims to have seen hysteria in cats. The cases of alleged hysteria in dogs reported by Mainzer (1906) have been criticized by Dexler, and the latter has shown that hysteria in animals occurs, at most seldom, or not at all, because it is prevented by the specifically animal mentality, by the inability to coordinate the relations of phenomena one to the other. Further, motor inhibition due to strong impression upon the senses, which was studied carefully by Verworn and which is quite frequent in animals, may be mistaken for motor disturbances that have an emotional basis. Vageler, among others, even assumed the existence of a supposedly imaginary pregnancy and took it also to be a sign of hysteria, but Kehrer explains these cases as being due to auto-intoxication with lutein which after an unfruitful oestrus is formed by the slowly retrogressing corpus luteum, and after its absorption produces an affection of the nerves which causes the milk glands to swell and secrete milk, so that the animal becomes restless and prepares for parturition.

Besides, the sexual perversity which is not uncommon in animals has been frequently attributed to a **degenerative psychopathic constitution** (psychic degeneration). Thus Cadiot relates that a 1½ year old dog used to play with the hens in the fowl yard, and developed the habit of covering one of the hens (whether inmissio penis occurred into the cloaca is not stated). A similar case was described by Villemain in a 10 months old dog, which had the habit of seizing a hen, holding its head fast with its mouth, and attempting to introduce his penis into her cloaca. The misused hens were killed by this violence; only one hen allowed the rape with resignation. Finally Holterbach noticed sexual intercourse between a bull and a mare. The last named author attributed the penetration of the vaginal wall of a cow by a powerful bull during covering to sadism. It would have to be proved, whether these and similar abnormalities of sexual life are actually to be considered as evidences of an abnormal psychic condition, for it is very easily possible that the perverse sexual intercourse is merely the result of a frequently agitated sexual desire arising from non-gratification of the normal sexual appetite, perhaps a kind of onanism or simply a phase of detumescence. In man also all onanists or pederasts can hardly be said to be mentally deranged or psychically degenerated, for the sexual perversity can without doubt be the result of psychoses, but it has often been observed independently of such a cause (Weygandt). Karsch and Lomer have given numerous examples of abnormalities of sexual life in otherwise healthy animals. In this connection the observation of Albrecht is also interesting where a dog with prostatitis which emitted an odor like trimethylamin, was jumped by other dogs or attracted them in like manner as if he were a bitch in heat.

Enzootic eretism in animals may be (see page 800) associated with the psychoses of man from congenital defects.

**Literature.** Albrecht, W. f. Tk., 1900, 161.—Dexler, *Ergebn. d. Path.*, 1900, VII, 401 (Lit.); *Monatschr. f. Psychol. u. Neurol.*, 1904, XVI, 99 (Lit.); *D. t. W.*, 1906, 525; 1908, 289; 1909, 61; *Neurol. Cbl.*, 1907, 98 (Lit.); *Die Tierpaniken*, A. f. Psych., 1907, XLII, 2 (Lit.); *Zur Diagnostik d. psychotischen Krankh. d. Haustiere*, Prager Med. Wochenschr., 1908-1909, XXXIII (Lit.).—Ebbinghaus, *Abriß d. Psychologie*, 1908.—Gleisberg, *Lehrb. d. vergl. Path.*, 1865.—Goldbeck, *D. t. W.*, 1902; 201.—Grobon, *Rev. vét.*, 1907, 172.—Hoffmann, O. M., 1899, 1.—Holterbach, *D. t. W.*, 1905, 519; *B. t. W.*, 1905, 217.—Karsch, *Päderastie u. Tribadie bei d. Tieren*, 1900 (Lit.).—Kehrer, *Die Umschau*, 1909, 171.—Lomer, *Neurol. Cbl.*, 1906, 513.—Mainzer, *ibid.*, 1906, 438.—Marchand, Basset & Pécard, *Rec.*, 1906, 813.—Straaten, *Maanedsskr.*, 1905, XVII, 1.—Vageler, *Die Umschau*, 1909, 157.—Verworn, *Die sog. Hypnose d. Tiere. Beitr. zur Physiol. d. Zentralnervensystems*, 1898.—Weston, *Rec.*, 1905, 180.—Ziehen, *Psychiatrie*, Leipzig, 1908.

# Diseases of the Organs of Locomotion

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## 1. Articular Rheumatism. *Rheumatismus Articulorum.*

(*Polyarthritidis Rheumatica.*)

Articular rheumatism is a febrile infectious disease in which several joints are attacked by a serous or sero-fibrinous inflammation at one time or one after the other.

As rheumatism (*ῥεύμα*, flux of the laity, because they believe that the disease material flows about in the body) acute inflammations of the serous membranes, synovial capsules, muscles and nerves have always been designated, which arise from cold, are accompanied by violent pains and usually attack several parts of the body at once or in turn. Recent investigations proved that most diseases called rheumatism result from infection, cold being at most a predisposing cause, and that the complaint often occurs without the intervention of cold. While the name is at present still used generally to denote certain muscular and joint affections, this is only done in order to indicate the manner in which the disease extends and also the frequent connection of the attack with catching cold, but a common cause of the diseases called rheumatism is no longer accepted.

**Occurrence.** The disease occurs relatively most frequently in cattle, very rarely in dogs (among 70,000 sick dogs Fröhner found only 92 cases), horses (Pfeiffer, Tetzner, Fröhner, Paneritius, Knabe), swine, goats and sheep; in the last two species of animals it was noticed as an enzootic (by Greswell in sheep, by Barthelémy in goats). Among cattle, mostly delicate good milkers become ill, oxen are affected much less often, and almost without exception only if they are kept in the barn for long periods, while grazing animals are scarcely ever affected.

**Etiology.** Improper keeping of the animals, especially feeding on watery fodder of no food value, appears to exercise a predisposing effect. A much more common influence in this direction is exercised by cold, damp air and draughts, especially if they strike the animal standing in a warm barn. Finally



all the external influences which cause chills and colds may be considered as causes. Consequently the illness is observed most frequently in the spring and autumn.

Cows may fall ill with symptoms of articular rheumatism a short time, 5 or 6 days, after calving. The disease occurs especially after calving or after the retention of the afterbirth.

The course of articular rheumatism and its close connection with various diseases which at present are known to be decidedly infectious (endocarditis, sero-fibrinous inflammation of the serous membranes) seem to point to the fact that the actual cause of joint rheumatism is some infection. In addition, it must be mentioned, that in connection with infectious diseases of certain organs (uterus, udder, and so on) an inflammation arises clinically, corresponding with articular rheumatism, concerning the infectious source of which there can be no doubt.

The view that articular rheumatism is an infectious disease receives still more support from the recent bacteriological investigations on sick persons (Guttman, Petron, Buday) which repeatedly gave positive results, and according to which the pus producing microorganisms, especially streptococci, play an important rôle in the production of articular rheumatism. Wassermann and Meyer express the same view, while von Strümpell found staphylococci as well, and Thiroloix & Rosenthal attributed the disease to the bac. *perfringens* var. *rheumatismi*. Considering the great similarity of the symptoms in animals to the disease in man bearing the same name, there can now scarcely be a doubt but that joint rheumatism of man is also caused by an infection.

In man articular rheumatism is usually preceded by catarrh of the pharynx. Meyer cultivated streptococci from the tonsils of persons suffering from articular rheumatism, and with their cultures he succeeded in producing not only a sero-hemorrhagic inflammation of the joints, but also an inflammation of certain serous membranes, and in some cases even a verrucous endocarditis. Gürich expressed it as his opinion that a connection between articular rheumatism and angina can no longer be disputed.

Lenhartz indeed claims that in man he has always found the exudate in diseased joints free from bacteria and considers those cases where streptococci are found in the joints as not belonging to the category of articular rheumatism. But the negative findings of Lenhartz do not argue against the infectious nature of articular rheumatism, since bacteria growing in any internal organ can exert an inflammatory effect in the joints through toxins circulating in the blood stream, or it may be that the bacteria imprisoned in the articular cavities disappear soon after the onset of inflammation.

The results of investigations in man, further the observation that cows fall ill with symptoms similar to articular rheumatism a short time after parturition, after an abortion, or if the afterbirth is retained, support the view that articular rheumatism in animals is, in most cases, if not in all, a secondary disease, in such manner that the infectious material itself or its toxins reach the joints from any, even slightly diseased, organ by way of the blood stream, possibly with the stimulation of predisposing causes, and set up an inflammatory process therein. This view is strengthened all the more by a case of typical articular rheumatism which was recently observed by Knabe and which had developed in association with a pharyngitis. There is therefore no ground for separating diseases similar to articular rheumatism from this disease in so far as they do not represent the partial symptoms of specific infectious disease. Such affections are inflammation of joints, arising by metastasis after parturition, in connection with a mastitis, or with other internal disease. Yet many authors (Hess, Guillebeau, Ehrhardt, Strebel, Moussu, Leblanc & Bitard and others) still deem such a separation necessary. It would not have a proper basis even if it



are the stifle, carpal, hock and fetlock joints. A prominent symptom is acute pain, and as a result the animal goes lame and resents the joint being handled, seeking to avoid its being subjected to passive movement. On standing at rest the affected extremity will be bent (Fig. 117) and in case several feet are affected at the same time the animal remains lying down. The diseased joint and its neighborhood are swollen, hot and tense, in severe cases fluctuating in places. The local symptoms of inflammation become milder after a certain time, generally after one or two weeks; they may also disappear within a very short time, but frequently reappear later on in other joints. In this way the inflammation may attack most of the joints of the extremities in succession, while the other joints are affected only exceptionally; at times, however, the same joint is attacked repeatedly, and finally deformity of the joint occurs, whereby its mobility suffers an increasing restriction.

At the commencement there occurs a febrile rise of temperature, and at times the symptoms of fever precede the local symptoms.

The rise of temperature is usually considerable ( $40.5-41.0^{\circ}$  C.), and at the same time the breathing is quickened and shallow, while the number of pulse beats may be double the normal.

The appetite declines, rumination is suppressed, the discharge of feces is retarded, the urine is dark in color, its quantity is diminished. The milk secretion is likewise lowered or ceases altogether; the milk tastes sour and clots easily.

In animals that remain continuously lying down, pressure necrosis of the skin develops which may be the source of a general septic infection.

**Complications** may occur after a few days in severe cases, sometimes, however, they commence only during the stage of improvement. Most frequently inflammation of the serous and similar membranes is observed (serous inflammation of the tendons and tendon sheaths, less often verrucous or ulcerous endocarditis, further pericarditis, sero-fibrinous pleuritis, or peritonitis, etc.). The development of such symptoms is generally announced by a more decided rise of temperature and by general fever symptoms which are followed by the specific symptoms of the respective local affections.

**Course.** The separate attacks run an acute course and continue for 2 or 3 weeks; occasionally they last, with distinct remissions, for several months. The decline of an attack does not, however, mean a cure of the disease. On the contrary, it is very frequently noticed that a complete cessation of the symptoms occurs, followed after a certain time by a recurrence of the process in the same or in other joints, and that not only changes in the joint arise, but also the nutrition of the patient



suffers. At times catarrh of the stomach and indigestion develop, in consequence of which the animal rapidly becomes emaciated, the milk supply ceases, and the muscles corresponding to the severely affected joints, become atrophied. In this way a cachectic condition develops, which may, however, be due partially to certain sequelæ, especially inflammatory changes of other organs (valvular incompetence, growths on the serous membranes, etc.).

**Diagnosis.** The diagnosis of articular rheumatism necessitates great caution, since other diseases of the joints produce a more or less similar clinical picture.—Polyarthrititis puerperalis as well as joint inflammations sometimes following upon mastitis or upon inflammations of internal organs, which in many cases are limited to one joint, especially the hock joint, can be differentiated positively if the underlying illness can be determined, or if the complaint occurs shortly after parturition or abortion.—The other metastatic forms of the complaint in which frequently several joints are attacked by inflammation are also preceded by primary diseases of certain organs, and the arthritis in these cases is mostly purulent.—Traumatic inflammations are followed by feverish symptoms only after some time, and besides the history usually clears up the cause of the disease.

Arthritides arising in the course of different infectious diseases generally declare themselves only some time after the respective diseases have been in existence. In this respect tuberculous arthritis is not rarely an exception, since according to Guillebeau it may arise without tuberculous disease of other organs, and on this account may often be mistaken for joint rheumatism. In tuberculous inflammations, however, in about four-fifths of the cases, only one joint is affected, especially the stifle joint, and tuberculosis of other organs may be established at least in many cases. At times the tuberculin test is serviceable, but it does not always prove absolutely that the arthritis is of a tuberculous nature.

In an advanced stage the disease may be mistaken for osteomalacia. In this disease, however, only the phalangeal joints are usually swollen, and further softness and brittleness of the bones will be noticed; moreover, in large cattle herds the symptoms of licking or gnawing disease are observed.—In rachitis one finds only the articular ends of the long bones swollen in addition to rachitic changes in the bony framework; the articular ends are hard as bone, and only in certain cases moderately sensitive to pressure, while the capsule as well as the cavity of the joint itself is unchanged.

**Prognosis.** This is generally unfavorable, because on the one hand fatal complications may set in, and on the other hand, repeated exacerbations greatly decrease the value of the ani-

mal, causing deformity of the joints as well as subsequent chronic disease. A cure rarely results from treatment and has been noticed most frequently in dogs and swine.

**Treatment.** For the acute inflammatory attacks salicylic acid with its salts, especially salicylate of soda, are said to be specifics, but now and then they fail, as a case of Fröhner proves. To large animals one may give 30 to 40 gm. 2 or 3 times daily, to small ones 1 to 2 gm. 3 or 4 times daily. (Mendel and Behr saw quicker effect in man after the intravenous injection of salicylic acid.) With this treatment the fever usually declines after the first day and the local symptoms also improve. In order to assure a favorable result it appears advisable to continue the treatment for a few days. In cases where salicylic acid does no good other remedies with a similar action such as antifebrin, antipyrin, salipyrin or salol may be given a trial.

For local treatment friction of the affected joints with iodoform, camphor, carbolic acid or gray mercury ointment may be administered, followed by warm or Priessnitz poultices or fomentations with camphorated Burow's solution. At the same time the animal must, of course, be kept at rest, stabled in a warm place and bedded on soft litter. If chronic changes occur they must be treated by massage and absorbents, if for any reason it seems desirable to keep the animal alive.—If the origin of the infection is discovered, this must, of course, be treated at the same time. Cattle are best slaughtered in good time, before they become emaciated.

Chenot reports very favorable results in three cases in the horse after paracentesis of the joint cavities with subsequent aspiration of the fluid exudate and injection of a 7 to 10% solution of sodium salicylate into the cavity of the joint.

**Literature.** Barthélemy, J. vét., 1894, 276.—Behr, Münch. m. W., 1904-1908.—Cadéac, Journ. vét., 1908, 24.—Chenot, Rec. d'hygiène et de méd. vét. mil., 1907, IX.—Dammann, Mag., 1871, 296.—Ehrhardt, Schw. A., 1896, XXXVIII, 122.—Fröhner, Monh., 1903, XIV, 448.—Guillebeau, Schw. A., 1898, XL, 1.—Gürich, Münch. m. W., 1904, 2089.—Harms, Hann. Jhb., 1871-72, 31.—Leblanc & Bitard, J. vét., 1900, 193.—Paracritius, Z. f. Vk., 1902, 389.—Pfeiffer, Monh., 1899, X, 155.—Strebel, Schw. A., 1903, XLV, 37.—Tetzner, Z. f. Vk., 1899, 53 (Lit.).—Webb, Journ. of comp. Path., 1908, XXX, 350.

**Other Polyarthritides.** Inflammations of joints clinically corresponding with articular rheumatism and more or less similar to it occur rather frequently in the domestic animals. Polyarthrititis puerperalis is one of the most frequent. It occurs as a serous, fibrinous, or purulent inflammation arising generally a short time after parturition, especially if the afterbirth has been retained; sometimes it does not occur until some time after a birth, when it is usually due to putrefactive material in the womb. Under symptoms of fever the hock joint is affected principally, and often at the same time the carpal joint; eventually also other joints are attacked either simultaneously or consecutively. The



disease often leads to emaciation, especially if several joints are affected or if an arthritis continues for a long time.

Quite similar symptoms of articular inflammation sometimes follow upon mastitis or diseases of internal organs.

In the course of certain specific infectious diseases, forms of polyarthritis may be noticed which simulate the acute or chronic forms of joint rheumatism. Diseases leading to the development of such symptoms occur in fowl cholera, influenza of horses, distemper, swine erysipelas, hog cholera, foot-and-mouth disease and glanders. They usually appear as the disease declines, one or several joints being inflamed.

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**Infectious Articular Inflammation in Young Geese and Ducks.** (Socalled Lameness of Geese; Osteoarthritis infectiosa.) This affection, first observed by Prahl and described as "lameness of geese," was investigated by Lucet and its nature was recently cleared up by Freese. It occurs as an enzootic in geese and ducks 5 to 8 weeks old, and is caused by the staphylococcus pyogenes aureus. Lucet succeeded in producing artificial transmission of the disease in young geese by intravenous injection, and Freese caused it in like manner in young ducks.

The anatomical changes consist in a serous or sero-fibrinous inflammation of a joint, a hemorrhagic inflammation of the bone marrow, and in intestinal catarrh; in case of a protracted course the osteomyelitis assumes a purulent character.

**Symptoms.** Clinically the disease occurs in two forms.

In the acute form (peracute form of Lucet) one notices great apathy, complete loss of appetite, and besides severe lameness in one or both legs. The hock and single toe joints chiefly are affected, at times also single joints of the wings, especially the elbow joint; in the latter case the birds droop the affected wings. If the course of the disease is very rapid no further changes appear in the joints (Lucet); but in most cases one finds them also swollen, hot, painful and fluctuating. At the same time there is violent diarrhea, and often slight catarrh of the conjunctivæ. Death occurs within 2 to 4 days.

In the chronic form the symptoms of arthritis are most prominent, while the general symptoms are less pronounced, and diarrhea is noticed only at the beginning of the illness. After a duration of about 14 days death occurs, or the birds gradually recover, but they remain stunted and cannot be fattened. Exceptionally an acute relapse occurs which leads to death.

The treatment consists in the local employment of antiphlogistic or disinfectant remedies, but Prahl found these useless, while puncture of the joint cavity and subsequent fomentations with disinfectant solutions resulted in recovery of the animals in 12 days.

**Literature.** Freese, D. t. W., 1907, 322.—Lucet, A. P., 1892, 841.—Prahl, Pr. Mt., 1871-72, 168.

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**Wing Paralysis of Pigeons.** In carrier pigeons an arthritis sometimes occurs, also in consequence of infection, which assumes an enzootic character (Klee, personal observation). Housing in draughty, cold lofts predisposes to it.

It attacks the wing joints exclusively, and principally the elbow and shoulder joints, usually on one side, causing the bird to droop its wing and to be unable to fly. Now and then it is only by making the pigeons move that one can discern that one wing is hanging down. The affected joint is more or less swollen and painful.

For treatment Klee recommends putting on a woolen bandage and soaking it every two hours with lead water. By suitable bandaging and isolation, attempts at flying will be hindered and the joint kept at rest. After 8 to 14 days painting with tincture of iodine may be resorted to. A cure not infrequently results spontaneously. In a somewhat protracted course caseous masses form in the joint itself and in the neighboring tissues, which may be removed by operation; but when operated on the bird generally loses its ability to fly. (Klee, *Geflügel-Krankheiten*, 1905, 54.)

## 2. Muscular Rheumatism. *Rheumatismus musculorum*.

(*Myositis Rheumatica*.)

**Occurrence.** Muscular rheumatism is generally a rare disease, although now and then it may occur as an epizootic. There is no doubt that the disease is much rarer than was formerly supposed where it was often mistaken for other diseases (pachymeningitis spinalis; compression of the spinal cord, rachitis, osteomalacia, etc.). Horses and dogs suffer most frequently, yet the remaining domesticated animals may not infrequently be attacked. (Among the horses of the Prussian Army in the years 1899-1908 only 0.04 per cent of the whole stock on an average suffered from muscular rheumatism.)

**Etiology.** The important rôle played by cold cannot be disregarded, but perhaps here also it only acts as a predisposing cause, the immediate cause being probably an infection or intoxication. To support this view there is the similarity of the disease to articular rheumatism, as well as the fact that both diseases occur at times from the same cause and in the same animal. Magnin repeatedly noticed in horses attacks of affections similar to muscular rheumatism which developed after croupous pneumonia or pleuro-pneumonia, as well as in connection with an infectious illness which is not described exactly. The disease occurs, as a rule, after exposure to damp, cold air or draught, after sudden wetting through or after a cold bath, and is observed most frequently in the spring and autumn. In horses it arises further after long continued railway transportation as so-called pleurodynia (see page 84), in connection with hyperemia of the lungs, and perhaps also with fibrinous pleurisy (Sigl).

**Predisposition.** Horses and dogs seem most inclined to muscular rheumatism. Well nourished, pampered animals living most of the time in warm rooms are especially subject to it.

process is interfered with. Rather frequently the disease will jump from one part of the body to another, whereupon the form of the functional disturbances varies.

The sensibility of the skin remains unchanged as do also the reflexes, unless the voluntary fixation of certain joints hinders the releasing of tendon reflexes on account of the pain.

Fever is not usually present in muscular rheumatism; only in severe cases where a considerable part of the body is involved there is a rise of temperature of 1 to 1.5° C. On the contrary one finds the pulse almost always quickened and tense, and the breathing frequent and superficial. The appetite remains undiminished for the most part.

**Complications** occur very seldom; only exceptionally an acute inflammation of the serous membranes and catarrh of the respiratory or digestive tract follow upon muscular rheumatism, and in horses inflammation of the tendons, laminitis and possibly arthritis.

In foals Tátray observed acute iritis and choroiditis in about 5% of the severe cases with deposits of fibrinous exudate in the anterior chamber of the eye. In 90% of the sick animals the eye trouble disappeared within 6 to 8 days, in 10% attacks similar to periodic inflammation of the eyes were repeated and were finally followed by blindness.

**Course and Prognosis.** The disease usually runs an acute course and lasts only a few days or at most a week, whereupon the disturbances in motion disappear completely; sometimes, however, an inclination to relapses persists, so that the prognosis is somewhat less favorable, especially in horses.

Functional disturbances similar to those of muscular rheumatism may accompany inflammatory diseases of the tendons and fasciæ and the muscular sensitiveness may be found in strain, in overextension or in partial tearing of muscles.

**Diagnosis.** From all these complaints muscular rheumatism may be distinguished apart from the special signs noted in surgery, especially by its sudden occurrence, often after a chill, the firm consistency of the muscles, the gradual decrease of discomfort in movement and particularly the sudden transition of the complaint from one part of the body to another.—In contrast to tetanus only single groups of muscles are usually attacked in muscular rheumatism, a real trismus is wanting and the reflex irritability remains unchanged.—Paralytic hemoglobinemia is distinguished by severe derangement of motion, lack of pain in the muscles, loss of reflex movement as well as by the hemoglobin constituents of the urine.—Compression of the spinal cord and pachymeningitis spinalis were in former times often mistaken for chronic muscular rheumatism, the occurrence of which seems to be doubtful; but these diseases may easily be eliminated by a careful examination of the nervous



system, and the same applies to other nervous diseases.—Well defined cases of rachitis and osteomalacia may easily be distinguished, but where the disease of the bone is not pronounced the differential diagnosis is sometimes a matter of great difficulty.

**Treatment.** Muscular rheumatism limited to a small region is treated most suitably with massage, for which different stimulating materials (camphor, spirits of soap, mustard oil in alcohol) or chloroform (e. g. spir. sapon., chloroform aa, or spir. camph., spir. sapon. kalin. aa 100, ol. thereb. 20.) may be employed; after massage warm or Priessnitz poultices may be applied.

The production of diaphoresis is also of good service, and for this purpose animals should be wrapped up warmly, while dogs may be placed in a steam or turkish bath; subcutaneous injections of pilocarpine (0.2-0.5 gm.) may be employed, especially in horses.

In general muscular rheumatism salicylic acid and its preparations (see page 809) do good service. Salol, salipyrin, antipyrin, antifebrin and quinine (according to Caroni 20-40 gm. daily for a horse) are also effective. The combined employment of morphine and atropine in shoulder rheumatism of the horse may produce at times dangerous complications (see page 365). The disease of the eyes needs no special treatment.

**Literature.** Albrecht, W. f. Tk., 1902, 170.—Hink, D. t. W., 1899, 5.—Hoffmann, T. Z., 1901, 424.—Magnin, Rec., 1906, 217.—Schmid, W. f. Tk., 1901, 148.—Schwendimann, Schw. A., 1898, XL, 116.—Siedamgrotzky, S. B., 1874, 43; 1878, 41; 1887, 26.—Sigl, Monh., 1900, XI, 559.—Tátray, Vet., 1894, 209, 264.—Zschokke, Schw. A. 1898, XL, 97.

**Race-course Disease of Horses.** After excessive exertion, as for instance on a long run, stepping high, and on bad, deep, or hilly ground, after forced runs, in distance riding and on race-courses, perhaps also after being thrown, a peculiar diseased condition appears in horses which is commonly called race-course illness. It occurs mostly after work, but occasionally it may be noticed during the exercise.

The animals become feverish, exhibit great anxiety and seem to experience pain. Breathing appears labored and copious sweating occurs. The muscles of the thigh, back and neck are at first hard and stiff to the touch, and twitching as well as quivering of the muscles may be noticed; besides there is often retention of urine and of feces. After one or several days most of the above mentioned symptoms disappear, the animals begin to move about, but great weakness, increased irritability and timidity persist; the twitchings in different muscles are noticed for a long time and are increased by external influences. In the further course the stiffness of the muscles yields to a visible relaxation, the gait of the animal becoming remarkably languid and tottering, the feet scarcely being able to support the body. But these symptoms as well as the twitchings and the irritability disappear gradually, so that the animals recover completely in 3 to 4 weeks, and often earlier (Kirillow), but at times later (Wilhelm).

The basis of these disturbances is probably a myositis caused by overexertion or rupture of muscle fibers, although Wilhelm considers that there is a general disease of the motor nervous apparatus, and at the same time an inflammatory condition of the muscular tissue.

The treatment consists in complete rest, rubbing, massage, and stimulating applications.

**Literature.** Kirillow, *Vet. Jhb.*, 1891, 126.—Wilhelm, *S. B.*, 1897, 127.

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A muscular inflammation after over-exertion occurs comparatively frequently in cattle, and develops in the form of a serous inflammation, especially in the muscular tissue of the shoulder girdle (Marek). Pregnant cows and oxen which are stabled continuously are especially subject to it after long drives on foot. It does not appear to be a simple relaxation of tired muscle as Giovanoli claims; the affected muscles as well as the intermuscular connective tissue appear to be very much infiltrated with serum. The affected animals remain recumbent for 4 to 10 days or longer, are unable to rise on their fore feet, and if by chance they succeed in doing so there is a sinking of the vertebræ between the shoulder blades. Generally the complaint disappears of itself, but occasionally it necessitates slaughter. (Giovanoli, *Schw. A.*, 1909, LI, 116.—Marek, *Vet.* 1895, 308.)

**Muscle Degeneration.** A pronounced parenchymatous and fatty degeneration of muscles arises in the course of paralytic hemoglobinemias (see Vol. I). Besides cases have been recorded (Fröhner, Bartke, Leipziger, Cadéac and others) where the muscle degeneration in horses manifested itself after throwing, if the animals had struggled violently. The degeneration occurs in those muscles which come into play in the straining movements against the shackles, that is, the muscles of the loins and the croup, the anconeus and the extensors of the stifle.

This kind of muscle degeneration arises to all appearance in the same way as in paralytic hemoglobinemia, but there is usually no elimination of hemoglobin through the kidneys, although this symptom has at times been noted (Leipziger). Morel & Vieillard also referred the fatty degeneration as well as atrophy in the muscular tissue, which is frequently found in horses after being slaughtered, to previous attacks of paralytic hemoglobinemia. But it is not impossible that the degeneration of muscle may be combined with an inflammation caused by overexertion as occurred in cases of Fröhner and Cadéac.

**Literature.** Fröhner, *Monh.*, 1897, VIII, 499; 1898, IX, 489; 1899, X, 354.—Leipziger, *Z. f. Vk.*, 1900, 389.—Poulsen, *Maanedsskr.*, 1897, IX, 305.

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**Fatty Degeneration of the Muscles in Sucklings.** In fine-bred pigs and lambs, much less often in foals and calves, the muscular tissue of the whole body undergoes a high degree of fatty degeneration in consequence of anemia; the cause of this degeneration is as yet unknown (hereditary predisposition, too close breeding, excessively high fat contents of the milk). The muscles appear glistening like bacon, or as if cooked, and a fatty degeneration of the parenchymatous organs and the lymphatic glands may be recognized. The animals are born diseased (Fürstenberg, Roloff, Repiquet) or the degeneration may be seen a few weeks after birth.

The sick animals cease to suck, they are weak, scarcely move about at all, lie on the ground almost uninterruptedly (little pigs do not squeal) and die quietly or after the onset of diarrhea, and perhaps in convulsions.

According to the investigations of Tempel and others, trichinæ occur in some neighborhoods to a greater extent in slaughtered dogs (in four years on an average 1.11%), than in swine (of 289 slaughtered dogs Tempel found four with trichinæ [1.4%]). In the year 1906 trichinosis was found in 0.222% of slaughtered dogs.

In swine imported from Hungary into Saxony trichinæ were found in 0.011% in 1893; in 1894, 0.009%; and in 1895, 0.024% (Ostertag); recently swine imported from Austro-Hungary have been found to be more subject to trichinæ than the native breeds (Edelmann).

Trichinosis is much more frequent in North America. Thus Billings found 4% and Salmon 2.7% of swine in Boston to be affected with trichinæ. In the year 1874 the percentage in Indiana was 16.3, later 6.5; in Chicago, in the year 1878, 8%; in 1883, 2.4%; in 1881, in New Orleans, 0.4%. According to Hamel-Roos, in Boston, from 1886-1890, out of 3,064 swine examined, the boars were infected in 14.87% of cases, and the sows in 10.61%. Of American hams and other pig products imported into Germany, an average of 2.3% (maximum 8%) were found to be trichinous (Friedberger & Fröhner).

**Etiology.** The *Trichina spiralis* (*Trichinella spiralis*) is a small nematode belonging to the family of Trichotrachelides (see page 487) which occurs in the body of susceptible animals and of man and causes the disease. It occurs in a sexually mature state in the intestinal canal, and as larvæ in the muscular tissue.

The intestinal trichina is barely visible to the naked eye as a very fine, thread-like, yellowish white worm, whose straight anterior end becomes gradually thinner, while the blunt hind end appears bent. The surface of the body is flat with fine cross stripes; the male 1.4 to 1.6 mm. long, 0.04 mm. thick; the female 3 to 4 mm. long, 0.06 mm. thick. From the fertile eggs, with a diameter of 0.02 mm., embryos, about 0.12 mm. long, develop in the uterus of the female. They break through the eggshell in the uterus and are born alive. The male dies after copulation, but the female remains alive until after the birth of the embryos; her average duration of life is 5 to 8 weeks.

The muscle trichina at first lives free inside the sarcolemma of the muscle fibres, then rolls up and becomes enclosed in a lemon-shaped capsule. It is 0.7 to 1.0 mm. long, its head end forms a point, and its hind end is blunt.

The development of trichinae occurs as follows: After the eating of flesh containing the encapsulated, living muscle trichinæ the capsule is dissolved by the gastric juices within 18 to 20 hours, and the liberated larvæ reach the small intestine after 30 to 40 hours; they grow quickly, and in about 2½ days become sexually mature. Five days after copulation, after the female has been pressed into Lieberkühn's glands along with the male, she deposits living embryos in the interior of the glands. During a residence of 5 to 8 weeks a single female can produce, according to Braun, 8,000 to 10,000, and according to Neumann 15,000 embryos.

From Lieberkühn's glands the young trichinæ reach the lymphatics on the seventh day, according to the investigations of Cerfontaine, Geisse, Askanazy, and especially Graham; from there they pass to the thoracic duct and finally reach the blood stream, where they are deposited by the capillaries in different tissues, and there become arrested. Since the trichina embryo has been observed to have a spike-like process at the anterior of its body, Höyberg considers that it is capable of wandering actively from the intestine. As soon as the embryos reach the tissues of a convenient organ they develop there further if the conditions are favorable. The so-called wandering trichinæ going with the lymph stream into the lymphatic glands, and with the blood stream into the rest of the organs soon perish, but those reaching the striated muscles which contain sarcolemma persist. The wandering



Dogs which also frequently suffer from trichinosis infect themselves by eating rats which they catch. The same is true of polecats, while the infection of wild swine, foxes, badgers, hedgehogs, martens and marmots takes place by eating mice.

**Susceptibility.** In respect to susceptibility there is no difference between the various breeds of pigs. The spread of trichinosis, however, varies greatly in different neighborhoods, and in this respect the breeding and management of animals has a great influence. Infection through trichinous swine flesh and through rats occurs very easily where no great importance is attached to cleanliness and the burying of dead animals is not carried out immediately, where, further, parts of the bodies of dead swine are fed to others. At the same time there is more opportunity for infection where the swine are not reared in the open air and in large herds, but are kept in sties in the neighborhood of dwelling houses.

Many other animals are also susceptible to trichinosis, such as the wild hog, mice, dogs (Tempel found trichinæ in the flesh of slaughtered dogs as often as in that of swine), cats, wolves, bears, badgers, etc., horses, cows, sheep, rabbits, guinea pigs and hares can be infected artificially.

The susceptibility of different species of animals is not equally great. Mammals are the most susceptible, while in birds only intestinal trichinæ develop, but the young trichinæ soon disappear in the intestine. Genersich accounts for this difference by the fact that the chemical composition of the intestinal contents are always alkaline in rabbits, and frequently so in man, rats and mice, especially after hunger, while in dogs and birds (ducks) the intestinal contents show an acid reaction. It follows that the susceptibility of warm-blooded animals is in inverse ratio to the acid reaction of the intestinal contents.

**Anatomical Changes.** After the first week only a certain watery consistency of the muscular tissue is found; besides the cut surface appears somewhat cloudy, its color pale and transparent gray. From the fifth to the tenth week the muscle fibers, when cut in their long direction, show very fine gray or yellowish streaks, while somewhat later one sees very small yellowish gray, or grayish white little specks thickly clustered together or strewn about, which stand out sharply defined when examined against the light. The respiratory muscles such as the diaphragm and intercostal muscles are attacked most severely, also the muscles of the neck, larynx and tongue. (According to Böhm the muscles whose activity is the most intense are attacked most severely.) Within the muscles the trichinæ are found in greatest numbers in the superficially situated fibers and in the neighborhood of tendons, while the tendons themselves remain quite free from the parasites. In the heart muscle and in non-striated muscular tissue trichinæ never occur (no sarcolemma!).

For the certain recognition of trichinæ, microscopic examination is essential. According to Johne, this may be undertaken in fine oat-grain-sized pieces of muscle which, after the addition of water or common salt solution (0.7%), or acetic acid (0.1 to 0.5%), have been pressed together between two glass plates (the best form is the so-called compressorium). Ten, or at most 40 to 50 diameters of magnification are sufficient. By this means, free parasites or non-calcified capsules, enclosing young trichinæ, may be recognized very easily, while after calcification in a transparent light only the black, characteristic lemon-shaped capsules are visible. After addition of dilute hydrochloric acid the lime salts dissolve out, whereupon the trichinæ inside the capsule, which has now become transparent, appear in view. For examination the above named muscles are best adapted.

For scientific examination Tikhomiroff recommends the following procedure: The small-cut pieces of muscle that are to be examined are for half an hour put into a mixture of nitric acid (4 parts) and chloride of potassium (1 part), and then carefully shaken in distilled water, whereby the muscle is made to separate into fine fibres. Minute swellings may be perceived in the fibres with the naked eye; under the microscope the encapsuled trichinæ may easily be seen. Close puts a small piece of the suspected muscle in a conical glass containing a mixture of hydrochloric acid and pepsin. The separated trichinæ sink to the bottom and can easily be removed with the pipette.

As additional anatomical changes one finds, before encapsulation, only hyperemia of the lung and parenchymatous degeneration of the internal organs, further, acute intestinal catarrh and acute swelling of the mesenteric lymph glands, especially during the residence of trichinæ in the intestine.

Mature trichinæ are found 4 to 6 weeks, at latest 8 weeks, after infection in the anterior part of the small intestine, seldom in the large bowel. To discover these, the intestinal contents are diluted with water when the trichinæ may be recognized with the naked eye as very fine, short, whitish threads. Yet it is more suitable to put a drop of the mixture between the slide and cover glass and examine it with a magnification of 40 to 50 diameters.

**Symptoms.** In living swine trichinosis has not yet been diagnosed, although there is no doubt that after intensive infection symptoms of disease arise, but these are ascribed to other affections. The symptoms of trichinosis in swine have therefore been observed only after feeding experiments.

The disease of the intestine mostly declares itself 3 or 4 days after infection, at the latest towards the end of the first week. The animals suddenly become ailing, take no food, vomit at times, crouch in the straw and move about unwillingly with arched backs. Obstinate diarrhea is hardly ever absent; at first the feces are pultaceous, then watery and ill smelling, and the animals also show symptoms of colic. After an extensive infection the animals rapidly lose condition and sometimes die at the end of the second week, in other cases the symptoms of an attack of muscular disease manifest themselves.

The second stage generally sets in towards the end of the second week, and in the meantime the animal rubs itself on hard objects, moves about stiffly and later lies motionless in one place with outstretched or bent extremities. Breathing becomes very painful and superficial. Huskiness also is seldom absent. If the disease involves the muscles of mastication, chewing is hindered, and occasionally total trismus may arise, while the affection of the throat muscles causes difficulty in

swallowing. Some authors have also noticed circumscribed edema of the eyelids and feet. The temperature rises chiefly at the time of the migration of the trichinæ.

Recovery is the rule; only after a very intense infection is the result fatal. The symptoms continue on an average 4 to 6 weeks, that is, until the encapsulation of the young trichinæ, and then gradually cease. The encapsuled trichinæ, which are present in great numbers in the muscles, do not destroy the function of the muscles in any way, and the animals eat and grow afterwards just like healthy pigs.

In **dogs** the symptoms are quite similar to those of trichinosis in man (Dlugay). Dobbertin noticed severe eosinophilia in the blood, which had arisen at the cost of the neutrophile leucocytes and occurred at the beginning of the migration of the trichinæ; the migration had, however, outlasted it.

**Diagnosis.** It was stated previously that trichinosis by natural infection had hitherto never been diagnosed in living swine, since the symptoms are not always characteristic, and besides other diseases with similar symptoms occur. In this connection muscular rheumatism may be called to mind, but here no signs of a violent intestinal affection precede the muscular complaint. The diagnosis is facilitated if an infection can be shown to have occurred. At times the affected muscle may be harpooned or excised and may afford material for examination. Since sexually mature trichinæ occur in the feces of infected animals, the feces should be examined microscopically in suspicious cases.

**Prophylaxis.** The carcasses of swine dead of trichinosis should be destroyed by industrial processes, and the living animals housed in places from which rats and mice are far removed. In knackers' yards and slaughter houses the keeping of swine should be forbidden where infection may arise through offal and rats or mice. The annihilation of rats and mice is an important factor in avoidance of the disease.

**Literature.** Babes, Cbl. f. Bakt., 1906, XLII, 541.—Bahr, Z. f. Infkr., 1906, II, 62.—Dobbertin, Über d. Verhalten d. weissen Blutkörp. b. Hunde, usw., Diss., Leipzig, 1907.—Edelmann, Lehrb. d. Flhyg., 1907.—Höyberg, Z. f. Tm., 1907, XI, 299, 455; 1908, XII, 26.—Johne, Der Trichinenschauer, 1907.—Ostertag, Bibliographie d. Fleischbeschau, 1905 (complete Lit.); Fleischbeschau, 1904.—Ströse, Arb. d. G.-A. 1909, XXXIII, 109 (Lit.).

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**Trichinosis in Man.** Man is infected by the use of raw or underdone pork, or by dog flesh and, according to the number of trichinæ reaching his intestinal canal, either slightly or so severely that in certain endemics 30% of cases may end fatally. The symptoms are for the most part similar to those seen in experimentally infected animals. Here also the disease begins in the second half of the first week, at times earlier after infection, with acute catarrh of the stomach, the patient suffering from loss of appetite, nausea and diarrhea, accom-



panied by colicky pains and at times vomiting. At the time of the migration and encapsulation of young trichinæ one notices swelling, increased consistency and painfulness of the muscles, and accordingly superficial painful breathing, hoarseness of the voice, difficulty in swallowing and pain in masticating. More or less characteristic are the circumscribed edema of the eyelids and joints, severe perspiration, fever similar to that of typhoid fever, and finally lying with the extremities drawn up under the body. Hemorrhages into the mucous membrane and skin, prurigo, pustules, furuncles and severe perspiration may also be noticed. Mild cases last 3 to 6 weeks, severe ones for many months.

The diagnosis is supported by severe gastric disturbances which are followed by the peculiar muscular affection and by edema of the eyelids. It will further be strengthened by proofs of severe eosinophilia in the blood, which occurs at the earliest eight days after the use of trichinous flesh. The diagnosis becomes certain by the demonstration of trichinæ in an excised piece of muscle, or in the initial stage, in the blood; for this purpose the latter is mixed with acetic acid, centrifugized and stained according to Giemsa (Stäubli).

Endemics of trichinosis have occurred pretty frequently since the discovery of the parasite. Of a great number of outbreaks and cases of death the following may be related: at Hedersleben, in the years 1863 and 1864 out of 2,000 inhabitants, 337 cases of trichinosis and 16 deaths; at Linden, in the year 1874, 400 cases, 40 deaths; at Emersleben, in the year 1883, 403 cases, 66 deaths. Johns recorded in Saxony, between the years 1860 to 1889, no fewer than 109 endemics of trichinosis with 3,402 cases and 79 deaths. Since the year 1902 there have been annual endemics in Bavaria; in the year 1906 in Ingolstadt 8 cases; in the year 1908 in Rothenburg about 100 cases; in the year 1909 in Lorenzen 10 cases; in Markterbach and Wilhelmadorf about 50 cases. According to Friis the disease has been observed in Denmark on fifteen occasions. In Hungary the first outbreak was observed by Ballagi (1891) in the ironworks at Diósgyőr, where 26 persons were taken ill after eating sausages which came from Debreczen, and after suffering from one to four weeks all eventually recovered.

**Literature.** Merkel, Handb. d. ges. Therapie von Penzoldt-Stintzing, 1909, I, Bd., 353.

#### 4. Measles. Cysticercosis.

(*Ladrerie* [French]; *Finnenkrankheit* [German].)

**History.** Measles in swine was known in olden times to the Egyptians, Jews and Greeks, but it was first recognized as being due to a parasite by Hartmann in the year 1682. The connection of the disease with a tapeworm was suspected by Fabricius at the end of the eighteenth century, but the history of the development of the parasites was first recorded by van Beneden and Küchenmeister in the year 1850. Feeding experiments leading to like conclusions were further undertaken by Siebold, Haubner and Leuckart.

In cattle Leuckart produced measles artificially by feeding with the proglottides of *Tania saginata*, and similar results were obtained by Gurlt, Gerlach, Zürn, Küchenmeister, Leisering, Haubner; Hertwig, Ostertag, Breuer, etc., furnished further contributions to the knowledge of the disease in cattle.

**Occurrence.** The frequency of the disease stands in relation to the conditions of barns and houses in the affected re-

gions. Where the animals are kept near dwellings, about which bad hygienic conditions prevail, it occurs more frequently than where the collection and removal of excreta are well attended to. Consequently measles is generally much more frequent in the East than in the civilized countries of Europe where the frequency of cysticercosis has fallen to an inconsiderable percentage, especially in recent years. Swine are affected much more frequently than cattle, and the pigs of small farmers more frequently than the herds on large estates.

Measles was found in Germany, in the Kingdom of Prussia, in the years 1890 to 1895 on an average in 0.15% of 25,490,339 pigs examined; from 1897 to 1905 among 64,648,755 swine examined 0.05% were diseased; in 1904 to 1906 the percentages averaged 0.03, 0.025 and 0.04%. In the eastern provinces swine measles is much more frequent than in the western (Ostertag). In Saxony, in the years 1897 to 1902, the percentage varied between 0.01 and 0.04, the lower percentages applying to the last years; in 1906 a percentage of 0.01 was announced.

In Hungary, in the Budapest slaughter-house, in the years 1897 to 1901, out of 474,401 swine examined (many having come from Servia), an average of 1.08% were found to be measly. In the Budapest fattening establishment (Köbánya) Servian swine examined during life in the years 1895 to 1905 were found to be affected to the extent of 0.5% (v. Kukuljevič.)

**Cysticercosis of cattle** was found in the Kingdom of Prussia, in the years 1897 to 1901, in 0.55% of slaughtered cattle; in the year 1904 in 0.32%; in 1905 in 0.34%; and in the year 1906 in 0.37%. In Bavaria the percentage in the year 1906 was estimated at 0.06, and Zagelmeier found in the slaughter-house at Nürnberg 1.48% and 1.58% of cattle affected. In the Kingdom of Saxony, in the years 1897 to 1902, there was an average of 0.37% of slaughtered cattle diseased, whilst in the year 1906 the percentage was 0.22. In Augsburg, Stroh found measles in 0.04% of slaughtered calves.

In Hungary, Csáky and Breuer were the first to find cysticercosis in cattle. In Budapest, in the years 1897 to 1907, an average of 0.21 to 0.45% of slaughtered cattle were diseased, and in the last four years the percentages varied between 0.30 and 0.42.

In Switzerland, France and Italy cattle measles has also been noted, and especially so in Italy, and also in Tunis where, according to Alix, about 5% of the slaughtered cattle are found to be measly.

**Etiology.** Measles of swine is caused by the *Cysticercus cellulosæ*, the larval form of *Tænia solium*. This occurs as a cyst about the size of a lentil or small bean, round or oval in shape with a thin wall on whose internal surface a milk-white point may be seen; this is the invaginated scolex and is extruded on pressure upon the vesicle. Under the microscope one sees four suckers on the scolex, and between these a rostellum carrying 24 to 32 hooklets.

The measles of cattle is due to the *Cysticercus bovis* s. *inermis*, the larval form of *Tænia mediocanellata* s. *saginata*; it is similar to the cysticercus of swine, but it has a more longish oval form, appears somewhat reddish in color, and there is no rostellum on the scolex; one finds four suckers at the anterior end of the head, and a depression which corresponds to the rostellum.

The development of cysticerci occurs when proglottides, which get into the open with human feces, or eggs which have been liberated by the destruction of the proglottides, are taken up by swine or cattle. When the eggshell is dissolved by the digestive juices, the liberated oncospheres bore through the stomach wall and are carried by the

blood stream into different organs but especially into the muscular tissue. The onkospheres now change into simple vesicles or cysts, on whose wall small nodules arise later, and from these the heads of the future tapeworms develop.

The development occurs rather slowly. In 20 days the *Cysticercus cellulosus* is about the size of a pinhead and the head is visible as a small white point; after 40 days it appears as big as a mustard seed and the head may be plainly seen, but it has neither suckers nor hooks; after 60 days the cyst is as big as a pea, with suckers and hooks but no neck; after 3 months the bladder worm is fully developed and behind the scolex the transversely striped neck may be seen (Mooser). The growth of *Cysticercus bovis* occurs still more slowly. After 60 days the head appears as large as a pinhead with plainly visible suckers; the visible depression at the anterior end of the head develops in the sixth month, when the cyst has already reached the size of a small bean, but later it develops further and grows to a length of 12 mm. Hoefnagel & Reeser found cysticerci, 5 mm. in length, after 45 days, in a calf used for experimental infection.

**Natural infection** occurs by food or water which is contaminated by human feces containing the proglottides or liberated eggs, and is eaten by animals. Swine are attacked especially easily because they root about in dung heaps and in the neighborhood of latrines and sewage deposits, and because they root up soiled earth. Consequently the disease frequently occurs in such swine as move about freely in peasants' yards or in the neighborhood of sties built near dwelling houses, while the large herds which are mostly kept in a state of freedom are seldom attacked. The infection in cattle probably results primarily from food or drinking water contaminated by human fecal matter, but it is not eliminated on pastures if persons with tapeworm deposit their feces there, or if during floods the contents of canals have been carried to the pastures. Contamination of the drinking water is easy, as brooks frequently are found in the immediate neighborhood of latrines or manure heaps. It is also to be considered that eggs of the tapeworm are viable on damp ground for a very long time, and consequently even one infested man may infect the ground for a long time with the enormous number of deposited eggs (one proglottis contains about 30,000 eggs). (van Beneden found that eggs kept by him in alcohol for a year could still develop embryos.)

**Susceptibility.** Young animals under 2 years of age are especially susceptible. The disease does not occur in sucking animals, because generally no opportunity occurs for infection. Exceptionally, however, quite young animals fall ill; thus Deleidi and Noack found some caseated cysticerci in sucking calves under two months old and these must have been taken up shortly after birth. In these cases it was supposed that the infection had been caused through hens' eggs given to the calves by an attendant affected with tapeworm, whose hands were soiled. In older animals the toughness of the tissues is much less favorable for the migration and further growth of onkospheres.



**Anatomical Changes.** In the presence of cysticerci in the striated muscle of swine the muscles appear otherwise healthy. Only after very severe invasion the muscle is pale, reddish gray and soft, and the increased connective tissue is serously infiltrated. The number of cysts varies in different cases; thus Küchenmeister found in one case 133 in 17 grammes of flesh, and this number corresponds to 80,000 cysts in one kilogramme of muscle. The cysts are easily removable from the muscle and leave a visible cavity behind; when present in large numbers they are of different sizes and partly shrivelled or calcified. They occur most frequently in the neighborhood of the shoulder or in the deep muscles of the shoulder and chest, in the abdominal muscles, in the nape and neck muscles, in the diaphragm, in the intercostal muscles, and in the adductors of the thighs, further also in the muscles of tongue and heart; in more severe cases they may be found in the other muscles, further, in the brain, eyes, liver, spleen, lungs, lymphatic glands and in the fat.

In cattle the cysts occur chiefly in the internal and external muscles of mastication and in the heart muscle; more rarely in the muscular tissue of the tongue, of the neck and chest, and only exceptionally in the intestines. Their number is usually small, and only very seldom do cases occur where the muscular tissue contains numerous cysts throughout. (Hertwig once found 300 in half a pound of flesh.)

**Symptoms.** In swine cysticerci cause pronounced symptoms only after a very severe invasion or after localization in the eye or brain. The greater number of measly swine appear quite healthy. The disease usually is recognized only if an organ that can be examined directly is affected. Thus one finds the cysts now and then under the conjunctiva of the eye, on the bulbus, or on the inner surface of an eyelid in the form of a bluish, transparent elevation. Sometimes, however, they are situated in the interior of the eye, in the anterior chamber of the eye, in the lens, where they can be recognized with the naked eye. But if they localize behind the lens, as happens rarely, they can be seen only with the ophthalmoscope. In the tongue cysts are to be felt with the hand, mostly at the edges, on the under surface or in the frænum. If the cysticerci are numerous, the tongue may be almost paralyzed, and in consequence the animal is unable to eat (Sobotta). Much more rarely cysts occur under the mucous membrane of the anus, where they may also be recognized by palpation.

In order to examine the tongue for cysticerci the pig is laid on one side by an attendant, and the mouth pried open with a stick. The veterinary surgeon passes the index and middle fingers of both hands into the mouth, and on drawing his fingers over the tongue and lingual ligaments, the cysts may be felt as firm nodules the size of a lentil or pea. The examination is of value only when a positive result is obtained. Small cicatrices in the above mentioned positions incline one to suspect that cysts have been cut out at some time. (A detailed description of the method of examination has been given by Kukuljević.)

afterwards small and somewhat painful nodes appeared under the skin, and these developed in three or four months to cysts as large as hens' eggs. They contained at first a reddish, and later on a sero-purulent fluid in which the cysticerci were suspended; later on the cysts broke spontaneously and fistulous openings occurred in their places. Among other symptoms, defecation, fever and loss of appetite were noticed.

In deer, measles (*Cysticercus cellulosæ*) have often been found during meat inspection (Borchmann, Agerth).

**Prophylaxis.** The occurrence of the disease in swine can be prevented by removing the animals far away from manure heaps, latrines, cesspools and drains. In the case of cattle the stalls must be kept clean, and the owners and attendants must be informed as to the nature and origin of the disease, and must be told not to deposit their feces in such places, in stalls (calf pens) or out in the open, where they will be accessible to domestic animals. The contents of privies should not be put on meadow and pasture land, but only on arable land that is not used for the growth of green fodder. A self evident prophylactic precaution is that persons affected with tapeworm should immediately submit to medical treatment.

**Literature.** Agerth, Z. f. Flhyg., 1906, XVI, 419.—Borchmann, *ibid.*, 1905, XV, 39.—Ciga, *ibid.*, 1905, XV, 118.—Deleidi, Clin. vet., 1903, 273.—Edelmann, Lehrb. d. Fleischbeschau, 1907.—Gunstow, Z. f. Flhyg., 1906, XVI, 419.—Hoefnagel & Reeser, Holl. Z., 1905, 351.—Kukuljevič, B. t. W., 1906, 626.—Noack, *ibid.*, 1906, 348.—Ostertag, Bibliographie d. Fleischbeschau, 1905 (complete Lit.); Fleisch beschau, 1904.—Öttle, Münch. t. W., 1909, 306.—Repiquet & Salvatori, J. vét., 1906, 220.—Schmidt, S. B., 1900, 270.—Stroh, Z. f. Flhyg., 1907, XVII, 78.—Suffran, Rev. vét., 1909, 401.—Trasbot, Dict., 1880, XI, 327.

**Sarcosporidiosis.** The order of sarcosporidia is represented only by a single species of sarcocyst whose young form of development is found in the muscle fibers, and constitutes the so-called Miescher's or psorosperm sacs. They occur especially in swine, sheep, horses, cattle and goats, and in swine most frequently in the abdominal muscles and diaphragm, in sheep in the skin and abdominal muscles, in horses in the neck and muscles of the throat. Miescher's sacs are always longitudinal in shape and contain numerous half-moon or kidney-shaped bodies (sporozoites); the protoplasm of the infested muscle fibers show uninjured striation. In case of calcification one still sees the kidney-shaped bodies around the calcified S-shaped formations. Their hard white capsule dissolves after the addition of potassium hydrate solution (which does not occur in the case of trichinæ). In their further development and growth the protoplasmic bodies of the muscle fibers disappear completely, so that the sarcosporidium is alone present in the widely dilated sarcolemma, and lies imbedded in the intra-muscular connective tissue. These little psorosperm sacs are very often encountered in the esophagus of sheep, goats, horses, buffaloes and deer. They seldom occur in the muscles of the larynx, chest or belly, or in the heart muscle. They appear as yellowish white cysts, of millet seed to hazelnut size, with pus-like contents.

There is no ground for separating the order of Sarcosporidia into the two classes of Miescheria and Balbiana depending on their presence in the muscle fibres or the intramuscular connective tissue respectively, since they only represent different forms of growth of the sarcocyst (Bertram, v. Ratz). There also occurs in the



horse the *Sarcocystis Bertrami*; in cattle and in buffaloes the *S. Blanchardi*; in sheep the *S. Tenella*, in the pig the *S. Miescheriana*, and in the hen the *S. Horvathi*.

**Infection** with sarcosporidiae occurs through the intestinal canal, as the investigations of Smith, M. Koch, Negre and Negri prove. Negri observed that the morphological peculiarities of the same kind of sarcosporidia differ according to the species of the experimental animal.

Sarcosporidiae were found in the muscular tissue of a llama (Rievel & Behrens) which contained a toxin having a paralytic action on the central nervous system, whilst Laveran & Mesnil have recovered a poison called sarcocystin from sarcosporidiae.

Lindner raises the question whether Miescher's sacs are not simply encysted vorticellary or colpidium protozoa which have been taken up with the water from pools. On the other hand Watson recognized a great similarity between the growing spores of sarcosporidia and the little bodies seen growing in trypanosomes, and considers that mistakes and failures in diagnosis may thus arise.

**Symptoms.** Sarcosporidia cause no disturbance of health except in the case of horses in which symptoms of illness due to sarcosporidiosis are often exhibited. Difficult respiration was noticed in a sheep and a goat (Dammann, v. Niederhäusern), in an ox stiff gait (Brouwier, Tokarenko), in a pig paralysis of the hind quarters (Virchow), in two pigs painfulness of the muscles, hoarseness, fever (Brzchosniowski), in a horse hardening of the tongue (Höflich) or of the tongue and the lips (Hendrickx & Liénaux), and as a result of this, nutritional disturbances. In one of these cases prominent nodes were found on the swollen, hardened tongue of a horse. Moussu & Coquot saw a hard diffuse swelling of the head in a horse similar to that seen in purpura, further urticaria-like swellings on the sides of the body, neck and under the chest, and also a diffuse swelling under the belly and on the sheath, as well as "wooden tongue" with small, yellowish red nodes on the under surface of the tongue. All these swellings were firm, of the consistence of cartilage, and situated under the skin; microscopic examination of an excised node showed the existence of sarcosporidiosis. The taking of food and water was made difficult by the changes caused, and movements were executed painfully and slowly. In another case noticed by Liénaux similar swellings were present, and lameness first in one leg, then another, and then in several legs; in the extirpated pieces of muscle sarcosporidia were found. Watson saw dejection, aimless wandering about with slow step, swelling of the bones of the skull. Sabrazès, Marchal & Muratet noticed fibrosarcoma-like swellings on the lower chest, and a considerable hard swelling of the metacarpal and metatarsal bones with formation of numerous exostoses, further, progressive anemia and emaciation leading to cachexia. In cattle with generalized sarcosporidiosis, Watson saw the following symptoms: Emaciation, frequent stretching of the head and neck with spasmodic quivering of the affected muscles, especially the muscles of mastication during the taking of food which was badly interfered with.

The **treatment** is not promising; a systematic trial might be given to potassium iodide, since Moussu & Coquot saw a lessening of the swellings and greater freedom of motion after its use.

**Literature.** Betegh, Cbl. f. Bakt., 1909, LII, Orig., 566.—Edelmann, Lehrb. d. Fleischbeschau, 1907.—Höflich, Münch. Jhb., 1896-97, 75.—Liénaux, Ann., 1907, 594.—Lindner, A. f. Tk., 1907, XXXIII, 432.—Michael, B. t. W., 1906, 619.—Moussu & Coquot, Bull., 1908, 445.—Negri, Cbl. f. Bakt., 1908, XLVII, Orig., 612.—Ostertag, Bibliographie d. Fleischbeschau (Lit.).—v. Rätz, Allattani, Közlemények, 1909, VIII, 1.—Rievel & Behrens, Cbl. f. Bakt., 1905, XXXV, Orig., 341.—Sabrazès, Marchal & Muratet, Rev. gén., 1910, XV, 177.—Watson, Journ. of comp. Path., 1909, XXII, 1.



# Diseases of the Skin

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## 1. Itching of the Skin. Pruritus Cutaneus.

(*Juckkrankheit.*)

An anomaly of function of otherwise apparently healthy skin which manifests itself by continuous or temporary itching of a more or less severe character is known as pruritus cutaneus.

This definition of itching does not apply to the cutaneous pruritus arising in diseases of the skin connected with anatomical changes (eczema, nettle-rash, etc.), or to the itching caused by animal or plant parasites.

Neither does the name apply, strictly speaking, to the paresthesia similar to itching which is seen now and again in certain diseases of nerves, especially in affections of the peripheral nerves.

**Etiology.** General itching of the skin is observed at times as a symptom accompanying chronic inflammation of the kidneys (Schindelka), chronic digestive ailments (Haubner, Schindelka) exceptionally also in jaundice, more frequently in diabetes melitus (Schindelka) as well as in shedding of the coat (Haubner). In one horse it arose after an attack of influenza (Pr. Mil. Vb.). Long continued feeding with maize and vetches as well as the prolonged use of caffeine may cause a general pruritus (Uebele). Further, according to Schindelka, it is not rare in marasmic dogs. In cases noticed in dogs by Eberhardt and by Fröhner there was no primary disease.

In opposition to the effect seen in human medicine, the prolonged use of arsenic seems to produce hyperesthesia of the skin rather than pruritus if the employment of the drug is interrupted periodically (Mayer).

Certain organic diseases cause a local itching of the skin. Thus pruritus ani is frequently due to the presence of intestinal worms (tapeworms, oxyuriasis, gadfly larvæ) in the rectum of horses and dogs, while nasal itching results from pentastomes and æstri. Finally Schindelka noticed pruritus of the point of the tail in dogs without visible cause, while Fröhner saw obstinate itching on the left side of the lower jaw in a horse.

Pruritus localis appears also as a symptom in certain paresthesias such as at the place of the bite in hydrophobia, in acute infectious bulbar paralysis at the

also in certain diseases of the brain, for example, after concussion of the brain (noted by Barbey in a dog). As a distinct disease it occurs very seldom. Hyperidrosis was noticed by Pott in horses after feeding on pumpkins.

Local or partial hyperidrosis also occurs chiefly as a secondary affection and is usually caused by traumatic lesions or by compression of the peripheral nerves or the sympatheticus, and more rarely by traumatic or inflammatory diseases of the spinal cord.

The known cases of partial hyperidrosis which have been reported so far, occurred as the result of traumatism at the neck (Dupas), front of the chest or



Fig. 118. Hyperidrosis localis in a horse. The dark stripe corresponds to the sweating intercostal region; at the upper end of the stripe there is a fracture of the rib.

shoulder region (Richter), also after fracture of the ilium (Delacroix), of the rib (personal observation), and after a trauma the nature of which is not stated (Dexler). Forgeot noted sweating appearing regularly after subcutaneous injections of ether, and limited to the point of injection; it lasted several days to three months. Local sweating occurs pretty frequently in inflammatory diseases of the pharyngeal region (Schindelka, Dexler, Dupas), while in one of Schindelka's cases, in a horse with pleuro-pneumonia, almost the whole of the anterior half of the body was affected. Compression of the peripheral nerves or of the sympatheticus through tumors or swellings now and again causes sweating (Kerlirzin, Dexler), while in certain cases of fractured vertebrae (Röder, Emshoff) the onset of girdle-like hyperidrosis commencing at the fracture has been noticed. Bielefeld saw a circumscribed sweating in the region of the forehead of a horse suffering from neuralgia of the trigeminus. Finally a case of unilateral hyperidrosis was noticed by Dexler in a horse with disseminated encephalo-myelitis.

of the skin. Alopecia is thus, as a matter of fact, the result of nutritional disturbances of the skin.

**Etiology.** Alopecia seldom occurs as an independent disease and has in this form been noted hitherto only in horses, dogs, and cattle where otherwise apparently healthy skin has been permanently denuded of hair in large patches (alopecia areata, Area Celsi). The symmetric occurrence of the affection may have a nervous cause or be due to some influence of the trophic nerves. Thus Joseph saw the affection in a characteristic form after section of the second nerve of the neck, while Trendelenburg noticed falling out of the feathers in pigeons after section of the sensory roots of the spinal cord. On the other hand in spite of negative microscopic findings, the action of parasites is not excluded with certainty.

Holborn found a fission fungus very similar to the *Tricophyton tonsurans*; Sabaroud believes that the falling out of the hair is caused by a *microbacillus* multiplying in the hair bulbs.

A **congenital baldness** (*Atrichia*, *Hypotrichia*, or incorrectly *Alopecia adnata*) is seen affecting the whole surface of the body in foals (Gherardi, Röder), calves (Laurent, Schaar), and dogs (Corniea). This condition occasionally stands in relation to a faulty growth of hoofs, claws and teeth (Bonnet), but many a time it is inherited. Such animals usually die after a short time, occasionally, however, the falling out of the hair only begins 1 or 2 years after birth.

André noticed general loss of hair in 11 foals of one mare, and in 8 of another the hair fell out, especially on the feet and on the lower parts of the trunk, near the second year of life. Koller saw a foal born without any hair, whose mother had lost her hair during pregnancy.

In by far the greater number of cases the falling out of the hair is a secondary affection (*alopecia symptomtica*). It occurs commonly in general derangement of nutrition, such as feeding with fodder of bad quality, when the animals are fed insufficiently, also as a result of intestinal catarrh (Duschanek) or of wasting diseases such as fluke, husk, etc.; on the other hand it may occur in the course of certain infectious diseases (purpura, strangles, influenza, swine plague and hog cholera), in which an injurious effect on the hair papillæ is probably produced by toxins circulating in the blood. It is more difficult to explain those cases in ewes, mares and bitches, where the wool or hair fall out at the end of pregnancy or during the lactation period (Hering, Spinola), also cases in horses after severe sweating and in individual horses as well as in large studs where during several succeeding winters or summers frequent cases of alopecia have occurred (Werner, Fomin, André). Ex-



ceptionally continual cold may, in horses, cause falling out of the hair (Hering, Kohlhepp). Wellach observed sudden baldness in young geese. In cage birds falling out of the feathers is often connected with the presence of blood-sucking parasites (*Dermanyssus*) in the neighborhood of the animals.

**Symptoms.** **Symptomatic alopecia** usually affects the whole body, at most the mane, tail and lower parts of the limbs escaping. At first one only has the impression of a more



Fig. 119. Alopecia Symptomtica.

active shedding of the coat, but later the hair coat is very much thinner, and eventually irregular spots become quite hairless; as these rapidly increase in size, the whole of the surface of the body becomes denuded and hairless (Fig. 119). In a portion of cases the falling out of the hair is preceded by edematous infiltration under the chest, under the belly and at the extremities, or by difficulties of digestion which may, however, ac-

company the process at a later stage. After a few weeks the points of fine little hair emerge from the denuded dark colored skin, causing the skin to feel downy, until finally the hair returns completely.

The general nutritional disturbances may in such cases become evident by the occurrence of transverse grooves on the matrix of horns, hoofs, claws and toes.

**Alopecia areata** appears in the form of small, scattered round spots on which the hair falls out, and which gradually increase in size, but for some time preserve a symmetrical round form, until finally large, irregular bald spots arise by coalescence of neighboring spots. The loss of hair may be extensive, but a general alopecia as in the before-mentioned cases, does not occur; on the other hand, the complaint is remarkably obstinate, and occasionally will not heal. The skin appears intact, and as a rule darker in color; at the edges of the spots the hair may easily be pulled out.

Röll & Siedamgrotzky found atrophic skin in their cases in the cells of the Malpighian layer, and very numerous pigmented bodies in the cells of the hair bulbs and sebaceous glands, while Schindelka found atrophy of the hair bulbs and of the fatty tissue and a diminution of pigment; microorganisms could not be found by him.

**Diagnosis.** Independent or congenital as well as secondary alopecia are distinguished by the healthy state of the skin, and by the great extent of the bald spots caused by tissue changes in the skin.

The round spots in alopecia areata appear very similar to those seen in herpes tonsurans, but in this latter one can often feel or see hair stumps, the surface of the skin is covered with fine scales and thread like fungi, and spores may be found in the hairs.—Not infrequently acariasis runs for some time a course similar to alopecia areata, but it may be differentiated by finding acarus mites under the microscope, and by the fact that serous fluid comes from the hair bulbs when a fold of the skin is pressed, although in many cases several examinations are necessary to establish the diagnosis. Alopecia due to acariasis occurs much more frequently in dogs than true alopecia, with which it has undoubtedly often been confounded.

**Treatment.** Since symptomatic general falling out of the hair heals as a rule of itself after a few weeks, the metabolism will most properly be stimulated by better nourishment, good care of the skin, by washing with soap, soap and alcohol rubs (pure rectified spirits, spirits of camphor). A similar procedure may be adopted in alopecia areata, but owing to the possibility of an infection, disinfectants (2% corrosive sublimate, 10% balsam of Peru, 5% pyrogallie acid) may be employed in addition to the removal of the loosened hair; as a matter of fact

even this treatment will prove successful only after a long time and sometimes not at all. Uebele claims to have obtained excellent results in making the hair grow by energetic daily applications by friction of nafalan.

**Literature.** Cieslik, B. t. W., 1906, 233.—Katske, Z. f. Vh., 1898, 289.—Kettner, *ibid.*, 1905, 122.—Koller, Rep., 1842, 306.—Krait, Rev. vét., 1905, 634.—Krüger, Z. f. Vh., 1893, 252.—Moussu, Rec., 1898, 81.—Röder, S. B., 1903, 296.—Rugg, Schw. A., 1890, XXX, 124.—Trendelenburg, Neurol. Cbl., 1906, 386.

**Trichorrhæxis Nodosa.** This affection which is not uncommon in the beard and hair of the head in man occurs also in horses, where it is seen generally as an enzootic. Exceptionally it may occur in swine, and Schindelka succeeded in observing the characteristic changes in the bristles of a shaving brush. On the tail and mane of horses, and occasionally also at one or several places on the hairs of the body, ball or spindle-shaped swellings occur in the hair which give it the appearance of being infested with white nits. Even on slight pulling the hair breaks at these places, and when the break is examined with the naked eye or better under a low power, a brush-like unravelling may be noticed. If many hairs close to one another are affected, and if the free ends are broken off during grooming, the hair becomes rough and it looks as if it had been singed; even at a distance the lustre of the affected spots is seen to be dulled; the tail may come to look like a rat tail. Contrary to what has been noticed hitherto, Bronec found in an enzootic, circumscribed swellings in the sheath of the hairs of the tail only, while on the rest of the body, chiefly in the mane and forelock, the tips of the hairs were split in the manner of a brush. The skin appeared rough, but itching was absent. The development always proceeds slowly and may require a year.

The cause of the disease is unknown at present. Medical men ascribe it to a disturbance of nutrition of the hair roots, or to intense rubbing of the skin, while Hodara asserts a parasitic cause, having found various shaped bacteria in the hair. The fact that many horses are affected, and the spread of the disease in a stud seem to indicate the infectious nature of the disease.

Steinhardt observed the spread of the infection from a few horses in a battery to almost all the horses of the regiment in 1 to 4 weeks; a similar observation was made by Schindler & Moser. In Schindelka's and Tennert's cases, men were infected. According to Schindelka the disease may be transmitted to cattle by inoculation. Jakob and Gross, however, did not succeed in proving the presence of micrococci in the hair.

The disease obstinately resists the different methods of treatment, but it disappears spontaneously after 3 or 4 months and then loses at the same time its infectious character; relapses, however, occur pretty frequently (Steinhardt).

For treatment it seems most suitable to clip the hair and to cleanse the skin thoroughly with disinfecting fluids. Trofimow found a mixture of oil of turpentine with tincture of cantharides most effective, Kalkoff a 5% watery solution of pyrogallol. Schindelka arrested the disease by clipping and washing the skin thoroughly, and then by long continued lotions of 1-2% sublimate solution, followed by sublimate compresses applied for one day. Bronec also succeeded with this method, but after drying the hair he rubbed in creolin and linseed oil.



Kutzner & Reichert recommend washing the skin with 2-3% lukewarm soda solution, and after it is dry, painting it with a 1% solution of pyoctaninum coeruleum. The last mentioned method was found effective by Schindler & Moser if a 3% solution was applied vigorously.

By way of prophylaxis the stables and curry combs, as well as the brushes should be disinfected.

**Literature.** Bronec, T. Z., 1908, 45.—Kalkoff, Z. f. Vk., 1892, 43; 1899, 263.—Kutzner & Reichert, *ibid.*, 1898, 223.—Römer, D. t. W., 1899, 216.—Schindelka, Hautkrkh., 1908, 532.—Schindler & Moser, Ö. M., 1906, 193.—Tennert, Z. f. Vk., 1902, 361.

#### 4. Dandruff. Seborrhoea.

The excessive formation of sebum arises, according to Schindelka, from a superficial inflammation of the skin with hypersecretion of the sebaceous glands, on which account fluid or oily secretions are excreted in considerable amount (seborrhoea oleosa) or scales form on the skin consisting of fat and the horny layer cells (seborrhoea sicca s. pityroides). The trouble may affect circumscribed places or develop all over the body (seborrhoea universalis).

**Occurrence.** These skin anomalies are chiefly observed in sheep, horses, asses, dogs, and at times also in cattle.

**Etiology.** The exact cause of the disease is not yet known. Only so much is known that anemic animals, or those suffering from internal diseases, are affected more frequently than healthy ones, and that not infrequently certain skin diseases (eczema, scab, acariasis, sclerodermia), severe infections, as well as digestive diseases form the basis for an attack of seborrhea. Very frequently no assignable cause can be discovered.

**Symptoms.** In horses the disease chiefly occurs on the neck, rump and face, but exceptionally the whole surface of the body may be attacked (Perrin). It occurs as seborrhoea sicca, and is also called exfoliating herpes or rash. In cleaning the animal mealy or bran-like scales are removed together with much hair. The coat appears dry, dull and as if filled with dust (pityriasis seborrhoica), but in addition bald spots form (alopecia seborrhoica s. furfuraceæ s. pityroides) which always increase very slowly. In other cases white, and later dirty gray, fatty, thick crusts form with a rancid smell, which in case of an extensive involvement emanates from the patients themselves (Röder). The skin appears either normal, reddened or sometimes eczematous.

In dogs seborrhoea sicca occurs mostly behind the ears, at the neck, on the shoulders and tail, whence it may spread to adjoining parts of the body. Aside from mealy or bran-like

scales, a greasy mass surrounds the origin of the hair shafts, which now and again mats several hairs into a tuft. Slight itching may at times be present, and eventually the hair will fall out.

Seborrhoea oleosa develops, as a rule, on parts of the body that are thickly covered with hair, but at times also in groin, inside the thighs or inside of the ears (Schindelka). The skin feels greasy or an oily substance may be scraped from it. The fatty substance, which is secreted copiously, dries to yellowish gray crusts under which the skin appears otherwise unchanged.

In sheep the neck, back, shoulders and sides of the breast are attacked mostly by seborrhoea sicca (so-called false scab), which form into small or large whitish yellow and later brownish yellow greasy scales or lamellæ (so-called tallow scab). Falling out of the wool occurs later. Itching does not occur, but the animals tear out the wool at the affected spot (Haubner).

Seborrhoea oleosa occurring on parts not covered with wool exhibits similar symptoms to those seen in the dog.

**Course.** The disease runs a rather slow course and unless treated does not improve except with some moderation in summer. Suitable treatment causes it to decline after the lapse of several months. In horses it sometimes leads to exhaustion of the patients.

**Diagnosis.** Seborrhea may be distinguished from eczema, scab or acariasis, because in these affections the skin itself is more or less changed, and scab is associated with evident itching. Scab and acariasis occur moreover often at first on distinct parts of the body, and on microscopic examination the mites are to be found there.

**Treatment.** Good nourishment, care of the skin, and if necessary, clipping of the hair, and the removal of any underlying disease, if such be discovered, usually lead to recovery. In dry seborrhea applications of vaseline or lanoline are indicated as well as washing with alkaline solutions or, in case of considerable itching, dressing with glycerinated salicylic acid (1 part of salicylic acid, 3 parts of glycerine, 60 parts of alcohol [Frederiks]). In other cases the disease is influenced favorably by the employment of a 5 to 10% resorcin or salicylic ointment. In obstinate cases the treatment must be continued for a month or over.

**Literature.** Dorst, Z. f. Vk., 1906, 437.—Moussu, Rec., 1898, 81.—Romi, D. t. W., 1904, 104 (Review).—Röder, S. B., 1903, 293.—Schindelka, Hautkrkh., 1908, 70 (Lit.).

## 5. Erythema.

By erythema is understood a reddening of the skin through a copious filling of the blood capillaries of the superficial layers

of the corium and especially of the papillary bodies, but without any important change of the tissue structure (*Erythema congestivum*) or at most with a slight serous infiltration of the skin (*E. exudativum*). The redness of the skin is thus, as a matter of fact, the first stage of an inflammation of the skin.

**Etiology.** **Primary erythema** (*E. idiopathicum*) develops under the influence of external irritants. The causes may be trauma, for instance, pressure, friction, blow, bite, etc. (*Erythema traumaticum*); thermic influences, such as scalding, burning, freezing, heat of the sun (*E. caloricum* and *E. solare*); also chemical agents, namely, sharp, irritating substances like vesicants (mustard, cantharides), volatile oils, mineral acids and lyes in not too strong concentration, further the irritating excretions of beetles and wasps (*E. toxicum*, s. *E. venenis ab acribus*). In new-born dogs and cats, Schindelka has noticed erythema lasting for several days (*E. neonatorum*).

**Secondary erythema** (*E. symptomaticum*) is seen chiefly in the course of general infectious diseases, as in swine erysipelas, swine plague and hog cholera, in distemper of the dog, in smallpox, in foot-and-mouth disease. In those cases in which erythema occurs after feeding on certain fodder, especially clover, buckwheat, distillers' grains, lucerne, a pathogenic effect on the cutaneous blood vessels is produced by a poison absorbed from the digestive tract.

**Symptoms.** Erythema is characterized by an increased redness of the skin, and for this reason the disease is recognized only in animals with unpigmented skin, namely, in sheep and swine, and also on uncolored, white-spotted parts of the skin of dark-haired or gray animals. The redness frequently is seen only in spots as large or somewhat larger than a lentil (*maculæ*, *roseolæ*) or in the form of larger congested spots which feel hot and at the same time are pink; in both cases the redness disappears under pressure from the finger but returns again soon. If serous infiltration exists, the affected part of the skin appears slightly swollen and eventually may show a yellowish color. In many cases the development of erythema is accompanied by itching, and then the affected parts of the skin are also slightly painful.

In most cases erythema lasts only a few hours or at most a few days, and then disappears without leaving a trace. If in exceptional cases it continues a longer time, the skin may desquamate as it becomes paler. If, however, the erythema occurs as the first symptom of an eruption or of a skin inflammation, then the respective symptoms (nodules, vesicles, etc.) usually develop as early as the second day.

**Treatment.** Interference is necessary in erythema only exceptionally, especially when it is accompanied by violent itch-



heat, and in another following upon a vaginal catarrh, while Houllier & Delannoy saw such cases follow the stoppage of the milk secretion, especially in market cows. Finally nettlerash may appear some hours after squeezing out the warbles in cattle affected with larvæ of the gadfly (Ströse). All these cases probably represent phenomena of hypersusceptibility to the absorption of heterogenous proteid matter, which gains access to the blood from the intestine in digestive disorders, or from other organs. Probably the homogenous proteid which has exuded into hollow organs acts like heterogenous proteid after undergoing certain changes.

Toxins of specific microorganisms are evidently of importance if the affection arises in the course of infectious diseases: for instance, in dourine (see Vol. I), in swine erysipelas, the mild form of which runs a course similar to nettlerash (Backsteinblattern) (see Vol. I), in purpura in which the edematous swellings sometimes develop from nettlerash; further, in influenza and in strangles where nettlerash is noticed rather frequently, especially in the convalescent stage; finally, it occurs in many animals after injections of mallein or tuberculin.

Both the primary as well as the secondary urticaria occur most frequently in horses, although cattle and swine may also be affected and more rarely dogs.

**Symptoms.** Nettlerash is sometimes preceded by indigestion, debility and a fever temperature (nettle fever, urticaria febrilis, febris urticata). Eggeling observed fever in cows up to 40.9° C, while feverish symptoms were observed by Perkuhn and Karpe in horses at the onset of the disease. The eruption occurs, however, mostly without these prodromal symptoms. Prominent swellings from pea to almond in size, hard, flat or half round, appear here and there on the skin, over which the hair appears somewhat ruffled. The swellings on the skin preserve these characteristics until they disappear, or they broaden on the surface and sink in at the center becoming ring shaped (*U. annularis*); by contact with one another several of these rings may form wave-like figures (*U. gyrata* s. *figurata*; seen especially well in horses suffering from dourine). On the skin of swine the swellings are at first of a reddish color, later they increase in size and fade in the middle (*U. porcellanea*), the progressing edge forming a red seam. If the affection is caused by the direct action of acrid substances, there exists in most cases a more or less intense itching, which otherwise is absent as a rule.

The development of the eruption may take place so quickly that within 5 to 30 minutes almost the whole surface of the body may be covered with elevations (Mecke, Jost). They last generally a very short time, a few hours or 1 to 2 days (in swine 4 to 6 days) and disappear without leaving a trace, after the swellings have become flattened (*U. ephemera*); occasion-

ally so much serum collects between the corium and the Malpighian layer that vesicles form (*U. vesiculosa et bullosa*, Pomphosis) which later on burst and heal with scab formation. It is also exceptional (e. g., in purpura) that urticaria attacks the mucous membranes of the eyes, nose, vagina or rectum; the elevations in the nose may temporarily hinder respiration. Now and then edematous swellings also arise at the same time. In general the disease is quite benign and assumes greater importance only if it often recurs in an animal (*U. chronica*, *Urticatio*).

In cows nettlerash develops occasionally owing to food of bad quality or after a sudden change of food. It is then usually very extensive, affecting the body, lips, wings of the nostrils and eyelids as well as the neighboring mucous membranes of the natural openings of



Fig. 120. Nettlerash.

the body and the larynx. In the neighborhood of the perineum knotty doughy swellings arise, which may hinder defecation more or less. While these swellings develop, the animals may be restless and constantly shift their weight from one foot to another. In consequence of the laryngeal involvement the animals may die of suffocation, while otherwise the attack rarely lasts more than 5 or 6 hours (Lucet, Tapken, Albrecht, Schleg, Wyssmann). Concerning the symptomatology of the mild form of erysipelas in swine see Vol. I.

In urticaria caused by stinging nettles one notices severe itching, and as a result restlessness or licking and rubbing of the affected parts of the body. In dogs severe rhinitis, stomatitis and pharyngitis may develop. Several dogs died from these affections. Holterbach believes that the cause of the fatal termination is found in the absorption of toxic substances, similar to snake venom, from the nettle hairs, and not in local inflammatory processes as is assumed by Rohr.



**Treatment.** Acute urticaria usually heals spontaneously within a short time and calls for no special therapeutic measures; still, fomentations with cold water or friction with vinegar or alcohol have a good effect. For the treatment of stinging nettle poisoning Holterbach recommends stimulating drugs (camphor) with permanganate of potash. If recurring attacks are the result of acrid substances, parasites etc., these must be



Fig. 121. Nettlerash in the dog, with diffuse swelling of the skin of the head.



Fig. 122. The same dog as in 121 after disappearance of the urticaria.

removed, while the presence of intestinal catarrh must be treated with aperients (neutral salts, castor oil) and disinfecting applications.

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**Serum Disease.** This phenomenon which sometimes occurs as a result of serum treatment has been known in human medicine since the introduction of the treatment with the diphtheria and the scarlet fever serums, and has recently been noticed also in animals, for example after the employment of anthrax immune serum in cattle and horses (Kovářzik, Zinner, Alexandrescu), of Gans's polyvalent pneumonia serum in two cows (Kovářzik), of Willerding's influenza serum in 30% of the inoculated horses (Bartels), and of septicidin in swine (Listo, Garaguso).

Essentially the serum disease consists in a hypersensitivity (anaphylaxis) of the animal organism to heterogeneous proteid substances. This hypersensitivity may be due to the fact either that the organism is especially intolerant of certain products of the dissociation of albumins, which may be a normal condition (idiosyncrasy; natural anaphylaxis of Detre), or that it contains certain substances which cause a rapid disintegration of the ingested proteid material lead-



ing to the formation of toxic products (artificial anaphylaxis of Detre). The natural hypersensibility shows considerable individual variations as regards the quantity and derivation of heterogenous proteid. Some anaphylactic animals are made ill only by considerable quantities of an alien proteid, while others react with severe symptoms of disease, even to small amounts of alien proteid (blood serum). The symptoms occur in all cases of natural anaphylaxis either immediately or soon after the ingestion of the respective proteids.

Artificial anaphylaxis, on the other hand, only develops subsequently to the introduction of certain proteid substances. It may indeed happen that the organism, being under the influence of some proteid (usually infectious substances) becomes intolerant to other heterogenous proteids just as in natural anaphylaxis; but usually, and in a typical manner, the hypersensibility develops after the animal has been treated with blood serum (serum anaphylaxis) or other heterogenous albumins. After a single injection under such circumstances the symptoms may occur only after 8 to 14 days (normal serum disease), after a sufficient amount of antibodies with the power of dissociating proteids have formed in the organism which was not originally hypersensitive, and these then cause a rapid disintegration of the heterogenous proteid substances which are still circulating in the blood. On the other hand, even very small amounts of serum will give rise to these symptoms only after one or several reinjections and then only in a portion of the animals. The symptoms either follow immediately (immediate reaction or serum disease) in cases in which the second injection was made after about 10 days, or only after 1 to 3 to 6 days if the interval was longer (hastened reaction). In the last two cases the anaphylaxis is strictly specific and occurs only in response to those substances with which the animal had been treated previously; it may also be transmitted passively to other animals, or by pregnant animals to the young (passive hypersensibility). Animals usually become anaphylactic primarily and exclusively in response to heterogenous proteids, but occasionally a homogenous proteid may give rise to serum disease as was shown by the observations of Lorenz and Bartels. In a case of the authors a healthy horse acquired serum disease after having been vaccinated with the serum of a healthy horse.

It may here be mentioned by the way, that it has been possible to render experimental animals anaphylactic (Rosenau, Anderson) by feeding blood serum or meat from healthy horses. This fact opens the way for an explanation of those cases of urticaria which occur occasionally after the ingestion of certain food stuffs.

The serum disease is only one form of anaphylaxis which includes among others the allergy of tuberculous or glanderous animals to tuberculin or mallein (see Vol. I).

**Symptoms.** The symptoms occur in domestic animals usually as soon as 5 to 30 minutes to 3 hours after a first serum injection; only Kovářík saw the occurrence of serum disease delayed until after the second injection which was administered seven days after the first. In horses there may be only a nettlerash with severe pruritus, which may involve the entire body (Bartels), or edematous swellings form, either alone or in association with the urticaria, affecting especially the head and the point of injection (Zinner). In cattle the most striking phenomena consist in a generalized urticaria associated with severe pruritus, and in edematous swellings which are sometimes severe and affect principally the head and the perineum. In addition one may observe great restlessness, pain at the point of injection, salivation,



trembling, generalized muscular weakness, dyspnea, bloating and occasionally a febrile rise in temperature. Not infrequently laryngeal edema may develop and may cause asphyxia. Some animals cough from the commencement and may continue to do so for weeks (Kovářzik). In hogs, Listo and Garaguso observed high fever, inappetence and very decided muscular weakness.

These symptoms are often severe; in domestic animals they usually disappear after a few hours or at most within a day without leaving a trace, a fatal outcome occurring only very rarely, mostly from asphyxiation. It is different in case of the anaphylaxis produced in small experiment animals by reinjections, in which the serum disease often ends with the death of the animal. In infected herds the disease may become important insofar as it diminishes the resistance of the organism to infectious substances for the time of its duration and may therefore give rise to the activation of some latent infectious disease.

**Treatment.** The treatment is similar to that employed in urticaria. The animals must be watched so that, in case of severe laryngeal edema, tracheotomy may be done in good time. In the employment of immune serums one should avoid wherever possible the use of heterogenous serum, and if the injections are to be repeated, this should be done at intervals of not more than 1 to 6 days or serums of different derivation should be injected; finally it is well to inject serums of as high a potency as possible so that comparatively small amounts of serum will suffice. The frequency and severity of the serum disease stand, other things being equal, in direct relation to the quantity of serum injected. The same serum has a stronger effect after intravenous than after subcutaneous injection.

**Literature.** Bartels, D. t. W., 1910, 485.—Detre, Á. L., 1910, 85.—Kovářzik, *ibid.*, 1909, 147, 563.—P. Th. Müller, Vorles. über Immun. und Infektion, Jena, 1909.—Wolff-Eisner, Klin. Immunitätslehre und Serodiagnostik, Jena, 1910.—Zinner, A. L., 1909, 636.

## 7. Eczema.

Eczema is an acute or, mostly, chronic inflammation of the papillary bodies and of the superficial layer of the corium of the skin associated with hyperemia, profuse exudation and itching. In the course of the inflammation, nodules, vesicles and pustules develop diffusely in the inflamed and swollen skin, leading to quick scaly excoriation of the integument and causing it to be moist and covered with crusts.

**Etiology.** The most frequent causes of eczema are found in external agencies acting on the skin. If the skin is neglected the dirt lodged between the hairs is balled, together with desquamated particles of epidermis upon the skin and begins to decay under the influence of dampness, the process being aided later on by the maceration of the corium. The quite juicy young epidermal cells of the Malpighian layer in this way come in direct contact with the decomposing scab, and are exposed to the influence of the schizomycetes present there, and also to the injurious action of the products of decomposition. Eczema



consequently occurs mostly in those parts of the body that are most subject to soiling, or which the animal is not able to clean thoroughly. Accordingly those parts of the body are affected principally which are covered with long hair, such as the tail and mane in horses where the dirt collects in large quantities at the roots of the hair, and on the back which is a favorite localization of the trouble in long-haired dogs.

Dampness also plays an important part, insofar as by softening its horny layer, it deprives the skin of its natural protection, and at the same time it promotes a firmer sticking of particles of dirt and the proliferation of fungi on the skin, and also a closer contact of the superficial with the deeper layers. For this reason the disease occurs in sheep after continuous rainy weather, affecting the back and croup, especially if the fleece is very close. In dogs eczema occasionally results from over-frequent washing, especially with green soap, which besides a loosening effect also exerts an irritating action on the skin. On the fetlocks of horses and cattle, pasturing on dew-covered meadows may give rise to eczema. Eczema (*Sudamina*) also develops in long continued diarrhea in the neighborhood of the anus, in dribbling of urine in the neighborhood of the urethral opening, further, on parts of the skin that sweat profusely, especially between the folds of thin skin; but in this case the excretions or secretions with their irritating constituents and products of decomposition are important factors. The continued effect of dampness and dirt must be held responsible for the eczema occurring on the fetlocks of horses and cattle, but seldom on those of sheep ("Mauke").

Mechanical influences, such as insect stings, rubbing, pressure, bites, may easily cause eczema if frequently repeated or if the skin sweats and is dirty. In the presence of animal parasites (flies, lice, ticks) not only the stings but also the factors already mentioned, and the prevailing uncleanness are to be considered as immediate causes, the same being true in the case of eczema accompanying scab and in eczema at the root of the tail in the presence of *oxyuris* eggs. In working animals, especially in horses, the disease occurs especially at those parts of the body which are subject to the pressure and the chafing of the harness or saddle, and here again constant intense sweating plays a part. The simultaneous effect of chafing and sweating may finally be seen in eczema occurring on parts of the skin that come in contact and rub on each other between the thighs, at the root of the tail, and in eczema occurring at times around the anus (*E. intertrigo*). The mechanical influences are further of importance in that they create a loss of substance in the epidermal layer of the skin, and render possible the penetration of microorganisms into the tissue of the cutis.

The influence of great heat may cause an eczema on the unpigmented portions of the skin (*E. caloricum*), and the same can develop on spots that are exposed for a long period to the hot



rays of the sun (*E. solare*), especially if the skin has previously been damp. In these cases the ultraviolet rays of the sunlight cause the inflammation.

Different chemical substances that cause loosening of the epidermal layer or irritate the skin may cause eczema, even if only used once. This may occur, especially after the application of gray mercury ointment (*Eczema mercuriale*), mustard oil, cantharides, croton oil, tartar emetic, iodine, balsams, petroleum, carbolic acid, tar, etc.; the affection also arises after the prolonged internal use of these drugs, and this is especially so in the case of mercurial eczema.

Of internal causes, diseases of the digestive organs are first to be considered (*E. symptomaticum*). Catarrhal diseases of the stomach and intestine especially, exert such an influence, for abnormal products of fermentation which are absorbed, probably affect the blood vessels of the skin or reduce its power of resistance to external injuries (autointoxication).

Weakening and exhausting diseases (fluke, verminous bronchitis, rickets, diabetes, etc.) cause a predisposition to eczematous affections. Other diseases of the skin, especially scab, also produce a decided predisposition to eczematous inflammation, but especially to moist eczema in the dog. Very fine skinned as well as old and fat animals, especially dogs, show a marked tendency to this disease, and individual and racial differences are evidently of importance in this respect (among dogs the pug dog, the poodle, the bull dog and the Leonberger [this is a breed of dog originally bred by Herr Burger of Leonberg, Württemberg. Trans.] are subject to the disease).

Schindelka repeatedly saw an eczematous skin disease, and finally nephritis, developing in consequence of constipation in the dog. He considers it probable that the toxins absorbed from the intestine irritate the skin on the one hand and the kidneys on the other.

The mode of origin and the course of eczema render it very probable that microorganisms, especially schizomycetes, are of importance. Hardly any experiments have been made in animals in this direction, and up to now those undertaken in humans have produced no conclusive result. Unna, who like Niemeyer, considers eczema to be a catarrh of the skin, found a species of cocci in the vesicles, scales and sections of skin, and also observed the development of eczematous patches after experimental inoculations with these cocci. Ravolgi found staphylococcus pyogenes albus, Bernheim the same coccus, and likewise the staph. citreus as well as the diplococcus albicans tardus; Scholtz found the st. citreus, Veiel found pyogenic staphylococci. According to the last named author the bacteria pass through the spaces between the epidermal cells into the skin and into the lymph spaces, and in this way cause the inflammatory process. Probably different kinds of bacteria exert a similar effect on the skin or on the tissue of the cutis. In one case of tail eczema in the horse, Casper proved the streptococcus pyogenes to be the cause of the disease; Baer on the contrary in eczema rubrum of a dog, found a micrococcus as the causal factor.

**Pathogenesis.** Eczema is essentially an inflammation of the superficial layers of the skin, in the course of which the blood vessels of the papillary bodies dilate and the non-pigmented

layer is markedly so, the sweat and fat glands become atrophic, and the skin, which has in the meantime become rough, is only capable of incomplete improvement (E. chronicum).

**Symptoms.** The changes sketched above run their course on the skin, and may, therefore, easily be watched on the living animal. In contrast to other skin diseases, one notices many variations in the morbid changes in their development, and especially a rapid development of papules and vesicles, as well as moist spots only covered by the deepest layer of cells, and formed by the papillæ, which come plainly into view after the crusts are removed. The recognition of the disease is also made easy by the fact that in some cases the different stages of development of the changes may be seen at the same time on the affected animals, and in this way the progress of development may be established by comparison, even without any history. On the other hand the little pathological formations easily escape because of the hair coat, and moreover severe inflammatory processes are caused by scratching, gnawing and rubbing, which are associated with hemorrhage and suppuration and affect not only the skin but also the subcutaneous connective tissue. This fact interferes with the clearness of the clinical picture.

Itching always accompanies this skin disease, and is the result of irritation of the nerve endings situated in the papillæ, which are subject to serous and cellular infiltration. It is especially intense in the acute cases as well as in extensive inflammation; it disturbs the animal considerably and is not dependent on the external temperature or on the time of day.

In acute cases fever sometimes arises, and a feverish exacerbation coincides with a fresh appearance of vesicles.

The great restlessness caused by the itching, the fever, as well as the long continued loss of albumin from the moist surfaces, considerably affect the condition of the animal in chronic cases, and in consequence this skin affection, which is benign of itself, may lead to considerable emaciation and even to complete exhaustion.

In the various species of animals the clinical picture of eczema is essentially the same, but in particular points deviations may be noticed which are important, both as regards the course and diagnosis of the affection, and which on this account will be mentioned separately, together with some etiological considerations.

#### (a) Eczema of the Horse.

In horses *eczema chronicum* (chronic squamous eczema, Kleien und Schuppenflechte, Hungerräude [German]; *eczéma sec* [French]) occurs particularly from insufficient nourishment and faulty skin hygiene, and develops in tender skinned,



anthemas, inflammations of the skin of the fetlock, which may correspond to erysipelatous, phlegmonous, or necrotic inflammations of the skin (Richter). Eczema itself, may, moreover, pass into these severe inflammations under the influence of mechanical factors. Richter asserts quite justly, therefore, that the unscientific collective name of "Mauke" should be dropped and the respective skin disease named according to its nature.

*Eczema of the bend of the carpal and tarsal joints* (Raspe) corresponds more to the previously described chronic eczema, and more frequently runs its course without complications; after the acute stage it assumes a scaly character, and acute attacks may subsequently arise in the neighborhood.

*Acute eczema of the extremities, of the lower part of the chest, of the lower belly, and of the inguinal region* arises through the influence of soaking and cold in horses after long continued work on muddy roads in cold, wet weather. On the skin of these parts which are splashed with the mud of the street, and especially on the inside of the extremities, an inflammation arises which bears the characteristics of acute eczema, and not infrequently edematous swellings may be noticed. As a result of the free exudation, scales and scabs form which mat the hair and later both fall off together. The changes disappear later on of their own accord.

*Chronic scurfy eczema of the long haired parts of the body* (socalled scurfiness of the mane and tail, plica polonica) develops on the upper part of the mane (socalled scurfy neck), also on the tail, and especially at the root of the tail (scurfy tail), rarely also on the head (socalled scurfy head and lips). Sweat and dirt predispose to its development, also gnawing and rubbing of the spots, caused by the itching; likewise, the fatty nature of these places and the thick layer of fat under the skin of the neck. Moreover, repeated washing with soap or frequent wetting promotes the occurrence of the disease by softening the horny layer of the skin.

The initial stage of the disease usually remains unnoticed since it affects badly cared for horses, and the skin is covered with long hair. Attention is first called to it when the animals become restless in consequence of the intense itching, and the hair begins to fall out. The hair of the mane becomes matted into a plait-like knot which can hardly be unraveled (Weichselzopf, plica polonica) from the accumulated dirt and secretion. After removal of the thick layer of dirt, which is usually only possible after clipping the hair, the greatly thickened skin which forms thick transverse wrinkles is brought to view, its moist and raw surface being covered with a smeary, sticky, fatty, ill-smelling mass composed of pus, epidermal detritus and dirt. At the tail the signs of acute inflammation, and especially the formation of pus are less in evidence, but the thickened skin is covered with dry scabs under which the skin shows the characteristics of eczema rubrum madidans, or on its surface only small dry scabs are found in place of crusts.

In some cases painful pustules arise in consequence of puru-



lent inflammation of the hair bulbs, from the middle of which a hair shaft emerges (eczema sycosiforme, Schindelka). In consequence of the proliferation of connective tissue in the corium and of its shrinking, the hair papillæ disappear, the fallen hairs are replaced by thinner ones, and later on no more hair grows. In this way the hair of the tail becomes thinner, until finally the root of the tail becomes quite bald, and only at the places situated lower down whisps of hair remain (socalled rat tail).

Seborrheic eczema (Eczema seborrhoicum, Unna) is met with occasionally in horses (Mégnin, Schindelka, Marek). The affection commences with the desquamation of many bran-like scales. Then lamellæ develop on the sides, on the nape of the



Fig. 123. Eczema Seborrhœicum.

neck and on the head, which are rounded or elliptoid in shape, as large as a dollar and up to 2 cm. in thickness; they are yellowish-gray or gray, have a greasy luster, feel greasy, and are depressed in the center. These lamellæ mat the hairs together and are easily removed with them. The skin is either only reddened (on the unpigmented parts of the body) especially around the outer margin of the deposits, or it is slightly moist. The disease may in time affect the entire surface of the body in which case the plaques, which are originally sharply circumscribed, may unite; it causes no or at most but little itching.

By suitable treatment it is easy to arrest the process for a short time, but relapses soon occur, so that the disease is hard to cure definitely, and in the meantime may lead to exhaustion of the patient.

The cause of seborrheic eczema is unknown, but the influence of microorganisms seems not improbable, although in a case of the authors it was not possible to transmit the disease to a healthy foal by rubbing the triturated scabs into the shaved and scarified skin, and in the cases hitherto observed no microorganisms were found. (In a case of the authors the mother of a diseased animal had apparently been similarly affected herself.)

To what class seborrheic eczema belongs has not yet been determined. Unna considers it an independent form of eczema which has a tendency to serpiginous extension as well as to central spontaneous healing, and is characterized by an abnormal high fat content of the deposition of the skin. The same author considers seborrheic eczema to be related to psoriasis, taking this to be only an extreme and extremely fatless modification of the very variable seborrheic eczema. On the other hand most medical men are not yet convinced that the eczematous nature of seborrheic eczema has been proved, and some consider it an open question whether it should not be looked upon as a trichophytic disease.—Schindelka considers the disease to be a form of eczema.

In donkeys Rossi noticed a seborrheic eczema. On the forehead, cheeks, neck, at the breast and under parts of the body the hair could easily be pulled out and was matted by orange yellow greasy scabs, the hairless places being covered with bran-like scales; intense itching was also present. Washing with warm lye and suitable feeding resulted in healing in a short time.

The exanthemata occurring in the course of strangles or catarrh of the air passages and often accompanied by itching (Cagny, Leblanc) should not be classified as an eczema, since it only constitutes a partial symptom of strangles (Compare Vol. I).

#### (b) Eczema of Cattle.

Apart from the eczema-like malt- and potato-eruption ("Schlempeausschlag") *acute eczema* occurs only rarely in cattle and develops chiefly in animals that are confined in dirty barns and are badly nourished and cared for. Favorite points of attack are the forehead, the back of the neck and the root of the tail, where it sometimes assumes the form of a squamous eczema, and again spreads over the whole surface of the skin causing moist eczema with intense itching. In the latter case pustules, and later on thick scaly scabs arise, so that now the character of the disease corresponds to that of eczema rubrum and impetiginosum. In this form the disease develops in the winter and especially in animals employed at hard work (Lafosse) and by constant rubbing deep seated inflammation of the skin and abscess formation may be produced.

On the other hand acute eczema may occur after the use of mercurial preparations, especially gray ointment (E. mercuriale) and in connection with scab. Eczema also occurs on the hind fetlocks, seldom on those of the fore limbs, and runs a similar course to that seen at the fold of the fetlocks in horses.

Between the digits an acute eczema (intertrigo) develops if the animals travel much on stony or muddy roads, or if, in the barn, their feet are constantly soiled by liquid manure. On the fine skin at this place little vesicles break out, which later change into intensely reddened, sensitive, moist spots.

At the end of the tail under the hair of its tuft an acute eczema (tail eczema) occurs in cattle which are continuously stabled. Dampness and dirt play a part in its production, especially if the animals are fed on sloppy food.

*Chronic eczema* can also develop, owing to want of clean-

**(c) Eczema in Sheep.**

*Chronic squamous eczema* in sheep occurs in the course of chronic diseases (fluke, verminous bronchitis, etc.) which lead to anemia and cachexia (socalled *Hungerräude*) and besides to desquamation of the dry and less elastic skin. A similar eczema also occurs in consequence of lack of attention and care.

The socalled greasy heel (*eczematous Mauke*) occurs on the fetlocks of sheep kept in damp and dirty places. It produces a form of disease similar to that of eczema of the fetlock in the horse.

Much more frequent and important is the acute, moist eczema (moist or fat scab, "*Regenfäule*"), which occurs in sheep in which the fleece is not very close on the back, and affects the back, loins and croup, but seldom the head, neck and shoulders. It is due to the animals getting wet through, especially in damp, cold weather, on the pasture. Under the influence of long continued dampness, the horny layer of the skin is loosened and an acute inflammation develops in the unprotected cutis, leaving a weeping red surface which is deprived of its horny layer and freely exudes serous fluid which dries and forms crusts. The skin is swollen, sensitive, cracked in places and the process is accompanied by more or less severe itching. The tufts of wool, which have become matted by the crusts, fall out, so that a considerable loss in wool may result. The sheep which are badly nourished are much debilitated if the affection persists for a long time. With the onset of dry weather, or after putting the sheep in clean roomy quarters, the skin dries up quickly and the inflammation disappears in a short time.

As *solar eczema*, an eczematous inflammation of the skin arises if the integument of the sheep is exposed to the hot rays of the sun immediately after shearing. Simultaneously with reddening, swelling and tenderness of the skin vesicles develop, followed by moist spots, and finally big crusts on the neck, back and sides. Young and feeble animals may succumb to the complaint.

*Intertrigo* is an eczema between the claws which is due to the same causes as in cattle (page 853).

**(d) Eczema of the Dog.**

Eczema undoubtedly is met with most frequently in dogs, and in a number of cases the various stages of its development may be followed quite readily. The form of the disease in individual cases is extremely variable. The inflammatory process is produced most frequently by faulty care of the skin, by dirt accumulated on the skin, parasitic fleas and lice, wetting of the hair in the neighborhood of the natural openings of the body, with excreta, etc.; the fine texture of the skin, especially in well-bred and fat dogs, and doubtlessly also digestive dis-



turbances exert a predisposing influence. In this connection strongly spiced or spoiled food has a prejudicial influence, as is proved by the fact that an emphatic improvement may be brought about in this case by internal treatment only.

The *acute diffuse eczema of thick-haired parts of the body* (eczema rubrum [Bär]) begins most frequently under the ears, on the neck, along the median line on the back, on the external surfaces of the thighs and on the shoulders. It declares itself by one or several occasional spots the size of a cent piece, which quickly increase in size, so that in one or two days they are as big as a tea plate and later even more extensive areas become affected (Fig. 125). The development of the eczema may be observed closely in all its stages often at the same place, from



Fig. 125. Eczema rubrum madidans with central encrustation in a dog.

the occurrence of redness of the skin, of papules and vesicles, to the pronounced swelling of the cuticle with moisture and scab formation; or the different stages may be observed in different parts of the body at one time. The severely diseased parts of the skin which usually exhibit roundish, sharply circumscribed, moist shining, orange yellow or red spots are surrounded by fresh papules and vesicles, and further outwards by a darker red ring. Between the papules and vesicles one also finds greatly swollen and glistening orifices of the hair bulbs. After a certain time the affected

skin is covered from the center outwards at first by a thin and yellowish-green crust, although it continues to secrete a serous or purulent exudate for some time. The process is always accompanied by great tenderness and very severe itching, which causes the animals to lick, rub, scratch and bite, and in consequence of this the inflammation extends into the deeper layer of the cutis and even into the subcutaneous connective tissue. In addition to a more intense swelling there occurs an exudate which is more purulent and which irritates the excoriated surface of the skin upon which many pustules and even abscesses may form; the hair falls out soon after the onset of the attack, or becomes thin at the periphery and stands upright. The regional lymphatic glands often show acute swelling.

The healing of the different eczematous patches usually occurs within 3 to 4 weeks or earlier, but owing to the recurrences which follow each other in rapid succession, the disease usually becomes protracted and changes into *chronic diffuse eczema*. In the course of this disease the skin becomes thickened and wrinkled, it cracks at places, bleeds easily, and the hair bulbs atrophy. In such cases complete recovery can scarcely be expected and, even in case of improvement, the skin remains thickened; scaling and desquamation continue on its dry surface, and here and there isolated hair grows which appears thin and wavy.

This disease is particularly frequent in long-haired dogs (at the Zürich clinic in the course of 4 years, 36% of the skin diseases of dogs were due to it); according to Bär, who has studied it closely, it is due to a micrococcus found by him in the affected skin, and he considers it a specific affection, having succeeded in two cases in producing the disease artificially with cultures of the micrococcus. A direct transmission of the disease does, however, never take place.

*Acute diffuse eczema* occurs much more rarely on the thinner parts of the skin and in such cases affects the groin and the inner surfaces of the thighs, extending later on to the belly and chest and still later to the flanks and elbow region, and on to the neighborhood of the lower lip, but it does not usually pass on to the parts of the body which are thickly covered with long hair (Mégnin). In this form eczema occurs most frequently in one or two-year-old dogs, and in certain breeds it is remarkably frequent, so that its occurrence appears to be favored by heredity (Cadéac; in Pokrowski's case, the whole of a litter became bald and suffered from eczema). The disease commences with the development of very small clear vesicles. Under the magnifying glass one can see numerous superficial, punctiform losses of substance, which later coalesce into a larger raw, exudating surface. Itching is intense, yet in spite of frequent scratching no scabs develop, nor are any severe changes produced. Suitable treatment rapidly leads to recovery.

*Seborrheic eczema* was observed by Schindelka, and was manifested by symptoms similar to those seen in the horse (see page 852).

*Circumscribed eczema* (*Eczema partiale*), in contrast to the other forms of eczema, shows no inclination to extend, and occurs in those parts of the body which are frequently exposed to mechanical irritation, macerated by secretions or irritated by dirt. The affection may either exhibit the characteristics of acute eczema or take a chronic course. In this form the circumscribed eczema frequently develops on the back (*E. dorsi*), especially in old animals and in insufficient care of the skin, and generally runs the course of chronic eczema. From the root of the tail to the withers, and not infrequently further forwards, the skin of the back appears bald or sparsely covered with hair;

it is usually unevenly thickened, forming thick and firm folds, and between these, as also in other places, it secretes moisture; sometimes scales and scabs are also encountered. The disease is accompanied from the commencement by intense itching, and sometimes the eczema is obstinate and persistent. Eczema occurring on the skin of the tip of the tail may lead to ulceration (tail ulcer) because the animal licks and gnaws the itching place.

Circumscribed eczema arises rather frequently on the bridge of the nose (*E. nasi*), on the cheeks (*E. buccarum*), on the arch of the orbit (*E. superciliorum*), and on the neck (*E. nuchæ*) where its occurrence may be due to wearing a muzzle or to the collar. In this form the lesion is generally moist and scaly, although small vesicles may be seen around the diseased parts, and on the neck, especially in fat animals, bleeding cracks between the folds of the skin. At the elbow joint or hock a chronic eczema may arise, which is evidently due to the irritation of lying or sitting on hard ground; it is characterized by the formation of thick, horny scales, accompanied by slight itching. In dogs the skin of the scrotum (*E. scroti*) or the prepuce, in bitches the lips of the vulva (*E. vulvæ*) may be the only localization of the disease, which is moist or vesicular on the scrotum of the dog, and may lead to severe swelling of the loose cellular subcutaneous tissue as a result of frequent rubbing of the fine skin. In the neighborhood of the other natural openings of the body the disease probably develops from the acrid action of the excreta; so especially on the eyelids (*E. palpebrarum*) in consequence of inflammation of the conjunctiva and of the lachrymal canal, on the lips, especially in the neighborhood of the angles of the mouth (*E. labiorum*) also about the anus and in the region of the perineum (*E. ani*).

Of the circumscribed forms of eczema, the greatest importance attaches to *eczema of the external ear and of the external auditory meatus*. This complaint is designated as inner ear-worm or otitis externa, and has been studied especially by Becker and Imhofer. Aside from the otitis externa, which develops in distemper as a painless moist eczema and accompanied by copious gray secretion (according to Lange in 50% of distemper cases [see Vol. I]), eczema of the external ear develops either at the same time with eczema of the other parts of the skin, or mostly as an independent affection through the influence of chemical irritants, although, at least in some of the cases, microorganisms, especially pus cocci, may be responsible. According to Imhofer the infectious material penetrates from the hair bulbs into the numerous glands of the auditory meatus and starts an inflammatory process which finally produces a connective tissue degeneration of the glands, in consequence of which, this disease should be distinguished from eczematous otitides and known as otitis externa genuina. Such a distinction cannot, however, be made clinically. In consequence of the



unfavorable anatomical conditions, ear wax and dirt easily accumulate in the external meatus, which then decompose and form irritant substances. The process of decomposition is favored by bandages on the head and the skin of the external meatus is now and then macerated by penetrating water or by medicines. The disease may also be produced by mechanical injuries, such as scratching, rubbing, violent shaking and the like, especially if animal parasites are present or if the meatus is soiled with substances which later decompose. Breed is of importance inasmuch as dogs with long and thick hair round the external meatus, such as dachshunds, setters and poodles, are chiefly attacked, as in them the ear wax does not soon dry up and soon decomposes because of the increased warmth in the auditory canal.

The symptoms consist in violent itching and severe pain, shaking the head, scratching of the ears, crying out or whining without apparent cause, and holding the head obliquely or stiffly, accordingly as one or both ears are affected. At the commencement or in a slight case the animals show pleasure when the ear is scratched or pressed, but later they avoid being touched; they even show an inclination to bite and appear surly and dejected. The external meatus usually contains a copious, yellowish, chocolate-colored to dark brown smeary ill-smelling secretion (otitis catarrhalis according to Becker) produced by a mixture of the serous exudate with the copiously secreted ceramen; sometimes it produces complete occlusion of the ear passage, and in consequence loss of hearing and matting of the hair around the ear. If pressure is exercised on the base of the ear, the secretions produce a squashing or popping noise. Sometimes one finds on the contrary merely a greenish yellow, evil-smelling pus without visible admixture of ear wax (otitis purulenta according to Becker). The skin itself appears diffusely reddened, swollen, sometimes cracked and easily bleeding, especially on the crests of its folds. Through long continuance the process becomes chronic, and leads to the formation of bleeding spots which may be as large as a cent piece. These show no tendency to heal, especially at the folds (otitis ulcerosa according to Becker), and if healing is effected, relapses occur readily. The complaint may be accompanied by slight to moderate fever.

In the meantime, a new formation of connective tissue takes place in the neighborhood of the ulcers, through which warty granulations form or more frequently a diffuse firm thickening of the whole auditory meatus occurs (otitis chronica hyperplastica according to Becker) and even lime salts may be deposited, as a result of which the ears may stand out from the head. At times the ear passage is completely closed through thickening of its wall, and then the few glands which have remained unaffected change to cyst-like cavities. As sequelæ of otitis may be noticed: othæmatoma, ulceration of the tip of

the ear, eczema or even phlegmons of the ear flap; in exceptional cases perforation of the drum of the ear and otitis media are said to occur.

A further form of circumscribed eczematous disease is the eczema of the skin between the toes (*E. interdigitale*, Intertrigo) which begins with a vesicular eruption, but later assumes a moist character, and in this form may continue for a long time; owing to the severe pain it greatly impedes locomotion. This form of eczema develops chiefly in pointers, probably as a result of irritation of the skin during the chase by thorns and punctures between the toes.

#### (e) Eczema of Swine.

In swine eczema is observed comparatively rarely and is designated as soot of young pigs (pitch scab, scab rash). It attacks almost exclusively only young pigs which are badly cared for and kept in dirty sties, or pigs which are weak and anemic from chronic diseases (rickets, swine plague, hog cholera, pyobacillosis). Sarcoptic scab often forms the foundation for the disease or the very frequent mange of young swine is mistaken for "soot of young pigs" or "pitch scab." On different parts of the body, but chiefly on the thighs, on the sides of the chest, on the belly, occasionally on the eyelids, an intensely itching vesicular eruption occurs, which now and then affects the whole surface of the body. The contents of the vesicles soon become purulent, but after bursting thick crusts form on the red moist surfaces which become dark brown or black in color because of the admixture of dirt. The affection, which is often mistaken for pox, exhausts the feeble animals greatly, but usually heals with suitable treatment.

In new-born litters of the higher breeds severe eczema-like skin disease, sometimes epizootic in extent, may develop on the second to fifth day of life. According to the description of Walthers a parchment-like covering develops at different places on the skin, on which small vesicles arise. After they burst blackish brown crusts, with central depressions, form on the sensitive and itching moist surfaces so that the skin appears as if smeared with soot. The sick animals are feverish, they suck little, become very weak, and finally die with symptoms of diarrhea. In 6 or 7 days all animals of a litter are affected, and the disease also attacks the litters of other sows as well as the teats of the mother swine. In strong animals the affection only develops 4 or 5 days after birth, and runs a mild course.

**Diagnosis.** Eczema is easily recognized in all species, when typically manifested, by the rapid development of nodules, vesicles and pustules as well as by the reddened and moist condition of the skin which in spots is deprived of the superficial layer of the epidermis. The last mentioned symptom makes it possible to distinguish between simple desquamation or scaling of the skin, because here the skin, still covered with horny epidermis, appears dry underneath the easily removable dry scales. The recognition of eczema is often rendered difficult

by the associated traumatic inflammation of the skin, because then the cutis is exposed through suppuration and tissue destruction, and even ulceration and purulent inflammation of the subcutaneous connective tissue may be present. In such cases it is possible to form an opinion on the nature of the disease from its locality and history; moreover, in eczema one often finds small vesicles and moist surfaces at the edges of the more severely inflamed parts of the skin. A special diagnostic significance attaches particularly to the presence of moist spots (Kaposi recommends in doubtful cases to rub diluted potassium hydrate on the affected skin, whereupon small moist dots or vesicles appear in cases of eczema). The more or less similar diseases with the formation of large crusts on the skin may be differentiated on this basis, since in them a deep necrotic ulcerous base becomes visible after removal of the crusts.

The demonstration of eczema does not, however, exhaust the requirements of diagnosis, for the discovery of the cause is of great importance, both practically, and from a therapeutic point of view. Skin diseases caused by animal parasites, and especially the different forms of scab frequently exhibit the characteristics of eczematous inflammation, and therefore a mistake in diagnosis is easily possible, especially in horses, dogs and swine. A slow development at the beginning and increased itching at night, and in warm places as well as the presence of vesicular eruptions speak in favor of a parasitic cause of the disease, but these signs cannot always be found at every examination even in the severe forms of eczema, and moreover in neglected cases only a severe form of dermatitis is generally present. Acariasis may be easily recognized by the favorite localization of the disease and in the pustulous form by the presence of large nodules and peculiar bluish transparent pustules as well as by the fact that the thickening of the skin is always considerable; the squamous form of acarus scab can in many cases only be differentiated from circumscribed eczema by the aid of the microscope, unless it occurs under the form of alopecia areata. Sarcoptic scab is often easily recognized on the basis of its occurrence in certain favorite spots and from the fact that in proportion to the severity of the skin disease the itching is intense, but not infrequently this also is only possible through microscopic examination and by demonstrating the transmissibility of the disease. Microscopic examination cannot be avoided, especially in those cases in which the customary treatment of eczema produces no improvement, since it is usually successful in cases that are not advanced too far. In suspected sarcoptic scab the examination often requires long and repeated investigation, while the dermatocoptes or dermatophagus mites are easier to find. Larger parasites which subsist and wander on the surface of the skin (ticks, fleas, lice) generally may be found without much difficulty.



In itching of the skin the integument at most shows only the effects of scratching, while changes in the skin itself are wanting. Acne in horses (so-called summer or heat rash, etc.) is distinguished from eczema by the occurrence of large, but not crowded, nodules between which the skin appears normal; at the same time sensitiveness to pressure is more pronounced than itching at the originally affected spots, and the disease heals in a short time. Dermatitis pustulosa contagiosa in contrast to eczema is characterized by its highly contagious nature and by the formation of small vesicles on roundish elevated spots of the skin, after their bursting gummy scabs form; itching is entirely absent. Erythema of the fetlock may be distinguished from eczema by its transient nature, but it may develop into eczema if neglected. Necrotic inflammation of the skin of the fetlock (so-called necrotic "greasy heels") may be differentiated from eczematous greasy heel by the character of its symptoms. Herpes tonsurans and favus may be distinguished from seborrheic eczema by their contagious nature, also by the presence of fungous elements round about the hair or in the superficial layers of the skin (sometimes difficult to prove), also by the fact that in herpes tonsurans the hairs break off closely at the orifice of the hair follicles or above, and in favus they appear dull and brittle, whilst in seborrheic eczema they fall out entirely; in favus also the peculiar character of the scabs is an important distinguishing factor.

Traumatic inflammations of the skin also enter into consideration in the diagnosis of eczema, but here the skin always undergoes a more or less deep destruction of tissue and a layer of skin of variable thickness mortifies, the inflammatory changes developing more by way of a reaction. Besides, suppuration is more frequent in this affection, while nodules, vesicles and pustules do not form.

Variola can only be mistaken for eczema in the initial stage or towards the end of the affection, otherwise the initial fever, the outbreak of the complaint over a large area of the body as well as the very characteristic appearance of the large vesicles should prevent error. The exanthema of distemper begins with the appearance of red spots followed by larger greenish colored flat pustules, after their rupture wrinkled dry deposits form; moisture is trivial and itching is, as a rule, completely absent. Foot-and-mouth disease may simulate intertrigo, yet its typical course, a similar eruption in the mouth and the contagious-infectious character of the complaint, especially on comparison of several cases of the disease, should furnish sufficient diagnostic points, besides the skin trouble is not so diffuse and uniform as in intertrigo.

Finally in those cases where the eczema is associated with anemia and cachexia, the cause of the bad nutrition must be investigated, besides a possible disease of the digestive organs

must always be borne in mind in view of the possibility of auto-intoxication

**Prognosis.** Eczema is generally a benign disease especially if its occurrence is attributable to external influences and if it is not very much neglected. In very chronic cases the deeper seated changes in the skin can, however, no longer be remedied, the more so as acute relapses frequently occur which again and again interrupt the improvement that has commenced to become manifest. In such badly neglected cases the sick animal falls away markedly in nutrition in consequence of continued unrest and loss of protein, and thus the prognosis is unfavorably influenced. Similarly the complaint may be regarded unfavorably if it occurs as a result of some chronic internal disease, which to a certain extent renders an energetic treatment of the skin disease impossible.

Relapses are observed frequently, and even in very mild cases and in those improving quickly under treatment a later attack is not precluded, although more frequently it occurs in badly nourished animals than in healthy ones.

**Treatment.** Above all the animals should be protected from certain external influences. Animal parasites should be destroyed by suitable remedies, dust and dirt should be removed with the brush. A great drawback to treatment, especially in advanced cases, is furnished by the hair coat, consequently it is very advisable to have the hair clipped at and about the diseased spots.

Itching must be controlled in every case in large animals by tying up and by covering, in small animals by well-constructed muzzles, collars or bandaging of the affected spots. Frequently the employment of even the most varied artifices does not achieve this object, until the alleviance of the itching or commencing healing in the inflammatory process secures relief to the animal.

In **acute eczema** it is advisable to protect the skin from the effects of water, soap or even air, and besides to remove the existing scales, crusts or secretions, which is most suitably accomplished with cotton soaked in oil, hydrogen peroxide, lime water, linseed oil, and if need be with compresses or poultices of 5% Burow's solution. For the last named object salicylic acid (as a 2 to 5% ointment or in the form of a 1 to 3% oil), or carbolized oil may be employed. In eczema of the ear warm creolin solution should be injected cautiously, and the hair of the neighborhood clipped, after which the collected secretions may be removed with forceps or a similar instrument, the end of which is wrapped with cotton soaked in oil or in creolin solution, and which is introduced as far as possible into the deepest parts of the auditory passage.

As long as only erythema, nodules and vesicles exist on the skin, drying or protecting substances, such as dusting powder or

ointment, may be applied. The best dusting powders are the strongly hygroscopic vegetable powders (Unna) because after absorbing the exuded secretion they turn it into a paste which protects the skin well. To these belong flour, *amylum tritici* and *a. oryzae*, lycopodium, tannic acid, etc. Very favorable results, however, are produced also by mineral substances such as zinc oxide, lead carbonate (white lead), talcum, *creta alba*, the different bismuth preparations (bismuth subnitrate, *airol*, dermatol, xeroform, thioform) iodoform, etc., which may be mixed advantageously with the vegetable powders in the proportion of 5-10:1. In more severe cases it seems proper to retain the thickly applied powder on the surface of the skin by means of a bandage, or to introduce it into the external auditory canal by shaking the flap of the ear or by insufflating the powder. On later treatments these powders are removed in the same manner as are the deposits on the skin. Besides the protecting powder, 10% creolin or ichthyol collodion, or traumatizin (guttapercha dissolved in 6 parts of chloroform) may be applied to those places on the skin that are suitable.

Ointments or pastes are properly employed later on or if dusting powders cannot be employed on account of long hair or will not stick to the skin. To these belong: zinc oxide ointment, carbonate of lead ointment, lead and tannic acid ointment, diachylon ointment with olive oil (Hebra) or with vaselin (Kaposi) or paraffin ( $\bar{a}\bar{a}$ ); the ung. simplex (Unna), in horses hydrarg. bichlor. ammoniatum, which often has a very good effect (1 part to 10 parts of lard); Unna's zinc paste (10 parts of zinc oxide, 2 parts of terra silica, 28 parts of benzoated lard) or Unna's sulphur-zinc paste (4 parts of precipitated sulphur to the previously mentioned paste). Just as effective are sapolan, 5 to 10% protargol ointment, also nafalan (according to Uebele best as house nafalan; 50% nafalan, 15% zinc oxide, 20% *adepts lanae anhydr.*, 15% solid paraffin), naftalan as well as pastes made with *airol*, xeroform, iodoform. Reinhardt found a mixture of 15 parts of crude carbolic acid and 100 parts of green soap very efficacious in eczema of the fold of the fetlock; this was spread over the skin and a bandage applied.

Itching is usually relieved or allayed entirely by means of the dusting powders or ointments; but if in spite of these it is very severe, good service may be afforded by Jessner's ichthyol paste (ichthyol, zinc oxide and powdered starch  $\bar{a}\bar{a}$  1 part, vaseline 2 parts), possibly painting with 10% lunar caustic, or compresses of lead water, Goulard's extract, or Burow's solution may be employed. In case of need the following may be suitably employed: anesthesin in 3% alcoholic solution or as a 10% ointment, or as dusting powder with 9 parts of *pulvis salicylicus cum talco*, also cocaine ointment (1 part cocaine with 25 parts of paraffin ointment or lime water-linseed oil). Schlesinger reports quick and favorable results with dymal (*didymum salicylicum*) used as a dusting powder or as a 10% ointment.



In the presence of much exudation the drying powders mentioned above have a good effect (ointments do not adhere to the moist surfaces!). After cleaning the diseased surfaces the powder should be applied thickly 2 or 3 times daily and before each application the skin should be cleansed of the adherent powder by means of oil. Since dusting powders are apt to form crumbly masses in the presence of copious secretion and thus irritate the skin, their employment must at times be discontinued and thick pastes be substituted. Thiol is suitable as a dusting powder or as a solution in water and glycerine (2:5:5), while Bissauge employs powdered sugar with good result. In more severe cases painting the part with 2 to 6% silver nitrate or picric acid (1 part to 86 parts of water [Lassartesse]) and subsequent application of powders are usually efficient, but this expedient cannot always be resorted to in light-haired horses, because the remedies stain the hairs dark or yellow. All the remedies indicated above dry the surface of the skin, while silver nitrate or picric acid coagulate the serous exudate, thus hardening the superficial layers of the epidermis. As soon as the secretion of moisture becomes less, ointments or pastes may be applied.

The skin should be protected from external influences for some time after it has become covered with fresh epithelium. For this purpose boracic acid (2-3%) mixed with vaseline is always useful, further Unna's zinc glue (white glue and glycerine of each 20 parts, zinc oxide 60 parts, water 100 parts) as well as nafalan, naftalan and safolan.

In the treatment of **chronic** and **seborrheic eczema** one must begin by removing the large crusts in the manner already indicated as well as by cutting away the warty growths or skin excrescences which may have formed; in most cases washing with soap may be employed, and aside from seborrheic eczema it seems suitable to make fatty applications. The further treatment is determined by the condition of the surface of the skin or by the state of the epidermis. If moist surfaces or folliculitis are present, the same means may be employed as in the moist stage of acute eczema or in acne (q. v.). If the exudation has ceased or folliculitis is relieved, or if this has not been present from the commencement, preparations of tar may be used advantageously, by means of which the separation of the large and cracked horny layer is facilitated. The different preparations of tar, such as *pix liquida*, *oleum rusci*, *ol. fagi*, *ol. cadinum* are all equally efficacious, and are generally employed diluted with oil, alcohol or fat. The following mixtures are in use among others: *pix liquida*, *sapo kalinus*, *spir. dilutus* 2:2:1; or *pix liquida*, *spir. dilutus*  $\bar{a}\bar{a}$ ; or *ol. cadinum*, *ol. sesami*  $\bar{a}\bar{a}$ ; or *ol. rusci*, *ol. fagi*, *ol. sesami*  $\bar{a}\bar{a}$ , etc. The liniment or oil is applied rather copiously on the diseased skin, left on for 3 to 5 days, and afterwards the black scab and scaly layer is carefully removed. The procedure can be repeated several times, but bearing in



mind the possibility of an intoxication, only small surfaces should be treated at one time. In order to avoid phenol poisoning Glauber's salts may be given in small doses internally. Creolin and lysol are effective and applied in the same form as tar. Still creolin should be used with caution, as contrary to the experience of Fröhner, the authors have often seen poisoning after its employment. In this stage good effects are also produced by naphthol and naphthaline (5 to 15% ointment [both of little use for the horse on account of danger from poisoning]), glycerinated iodine (1 part tincture of iodine and 4 parts glycerine), resorcin, salicylic acid (5 to 15% ointment), especially in marked thickenings of the horny layer or in seborrheic eczema. All these remedies cause a separation of the horny layer, lessen the itching and promote the absorption of the exudate from the tissue of the cutis. In obstinate chronic eczema of the horse with cracking of the skin, Röder saw good effects from sulphur and mercury ointment with the addition of cantharides, while Schindelka often obtained very good results in chronic seborrheic eczema of dogs by using sulphur baths and bran baths with the addition of sulphurated potash. In chronic greasy heel of the horse, Storch found lead nitrate efficacious when he used it after removing the granulomas from the cleansed skin, pressing it down with the finger in a layer as thick as the back of a knife blade and securing it with a bandage that was changed 3 or 4 times daily. Sommer healed very severe cases of eczema of the fetlock with Dealin (a combination of oxygen and fatty substances) in powder form.

If the disease process is already considerably improved and only few scales desquamate from the skin, which has regained its softness and elasticity, then the process of desquamation is hastened by rubbing in of bland remedies (olive oil, ungu. simplex). Any itching that may still be present at this stage may be controlled by nafalan, naftalan or sapolan.

In every kind of eczema internal treatment requires full consideration, especially if at the same time symptoms are present pointing to a disease of the digestive organs. In this connection the disinfection of the digestive canal or the employment of mild purgatives is proper. Thus Schindelka obtained good results in many cases in dogs through internal medicines (1.0 gm. of cinnamon powder, 1.5 gm. eucalyptol, 30 drops of oil of peppermint: 1.5-5 gm. daily in five doses). Arsenical preparations have always been ascribed a favorable influence (of Fowler's solution a tablespoonful for horses, 5 to 10 drops a day for dogs). This good influence is confirmed by recent observations. Paron and Urechie among others saw recovery occur in man after the internal use of calcium chlorate (3 gm. daily) in solution; improvement commenced the next day, and in five days the itching had disappeared. Care must be taken to secure the suitable feeding of the animal with non-irritating and easily digested food stuffs.



**Literature.** Bär, Schw. A., 1902, XLIV, 1.—Becker, Unters. üb. die Otitis. ext. d. Hundes. Diss. Giessen., 1907 (Lit.).—de Benedictis, Clin. Vet., 1904, 73.—Fröhner, Monh., 1908, XIX, 120, 124.—Imhofer, Beitr. z. Anat. etc. d. Ohres, d. Nase und d. Halses. Bd. II, 289 (Lit.).—Mathis, J. vet., 1901, 593.—Mouroux, Bull., 1904, 521.—Moussu. Rec., 1898, 81.—Noack, S. B., 1893, 123.—Prietsch, ibid., 1893, 124.—Qualdueci, Clin. vet., 1903, 281.—Richter, Z. f. Tm., 1905, IX, 23.—Roloff, Pr. Mt., 1868-69, 116.—Rottländer, Beitr. z. Ätiol. d. Ekzema in d. Fesselbeuge. Diss. Leipzig, 1908 (Lit.).—Siedamgrotzky, S. B., 1890, 21; 1892, 18.—Uana, Pathol. u. Therapie d. Ekzems. Wien, 1903.—Veiel, Münch. med. W., 1904, 1.—Walther, S. B., 1889, 79.

## 8. Food Rash.

### (a) Malt and Potato Eruption.

(*Schlempenmauke und Kartoffelausschlag, Rindermauke, Fussmauke, Fussgrind* [German]; *Eczéma des drèches de pommes de terre* [French].)

Malt eczema is a disease generally occurring only in cattle and affecting the skin of the ends of the extremities with a form of vesicular eczema. It is found mostly in steers and dry cows if they receive many potatoes or their industrial residues, especially distiller's slop for any length of time.

**History.** The disease was first described by Spinola in the year 1836. After him several authors investigated the nature of the affection, among others Roloff, Zürn, Rabe, Johne, Bräutigam, without, however, having solved the problem conclusively.

**Occurrence.** Malt eczema occurs chiefly in the fattening stables of potato distilleries, and especially in the spring. It causes considerable loss in the affected stock by preventing fattening and by occasional cases of death.

According to Ohlmann potato residues may also cause a similar eruption in horses; Gros-Claude and Frank saw the occurrence of an eruption in sucking foals whose mothers were fed with germinating potatoes or potato slop.

**Etiology.** The disease usually develops in those cattle which receive very little green and corn food and many potatoes. The effect is the same whether raw or cooked tubers, or leaves of the potato plant, or distiller's slop or other industrial residues of potatoes are used as food. Steers ingest an average of 60, cows of 40.1 liters of the potato slop daily without ill effects; 32 pounds of raw potatoes may cause a severe rash (Märker). These figures are not of absolute value, for in this respect not inconsiderable differences have been noticed in different years and at different times of the year in so far as, in spite of like methods of feeding, the disease is frequent in certain years and is moreover much more frequent in spring than during the rest of the year. That the disease occurs particularly in the spring is, at least in part, due to the circumstance



that the potato distilleries are generally in full operation only during the winter and the animals are generally fed only with distiller's slop at this time. Yet it appears not unlikely that the frequent occurrence of the affection in spring is related to the sprouting of the potatoes at that time or with their decomposition, or with the strong fermentation of the slop. Musterle has noticed that fermentation and acidification become very active if slop mixed with raw fodder is allowed to stand several hours before feeding. That this cannot be the only cause, however, is proved by the fact that even fresh tubers and the leaves or stalks of the potato plant can occasion the disease and even produce it in horses, if they are used as litter and come in contact with the skin of the fetlock region (Röll). This circumstance and the fact that malt eczema occurs only in certain years seem to prove that potatoes cause the disease only under certain conditions that are not yet clearly understood.

Concerning the **immediate cause** there seems to be a connection between the occurrence of the disease, the amount of slop or potatoes ingested and the presence of a chemical poison in the potatoes (Johne). The poison is absorbed from the digestive canal and gains entrance to the blood vessels of the skin, producing a similar effect on the papillary bodies and their epidermal layer to that of many mineral poisons (mercury, bromine). This view is supported by the relative immunity of milk cows, since these eliminate a great part of the poison through the milk, and if such milk is taken by sucking calves or foals a violent intestinal catarrh or a malt rash is caused in them. The preferable or even almost exclusive localization of the disease in the feet may be due to a mechanical irritation of these parts of the body and to the fact that they are most exposed to the action of liquid feces and soaked litter.

Solanin has been looked upon as the immediate cause of the disease, since this alkaloid is formed in great quantity in sprouting potatoes. But, apart from the absence of the characteristic nervous symptoms of solanin poisoning (depression, reeling, paralysis, retarded breathing), the correctness of this assumption is contradicted by the fact that the disease occurs also after feeding on solanin-free, sprouting potatoes as well as on the leafy portions of the plant.

In like manner the disease has been ascribed to bacterial toxins. Fission fungi may be stored up in the living plant and even more in the tubers and increase in time, producing a toxin, especially in the spring.

On the other hand the injurious effect of fatty acids and fusel oil has been assumed as a cause, yet the circumstance that slops of other origin also contain these substances and are not harmful, and that the complaint can be caused by raw potatoes or by potato leaves are against this view. Johné suspected the relatively large amount of potassium salts in the slop (1 liter of distiller's slop contained about 9.5 grammes of potassium). The symptoms of potassium poisoning (general muscular exhaustion and heart weakness) are, however, completely absent, and the idea of a dermatitis caused by potassium has hitherto not been supported by a single observation.

Zürn's hypothesis that the disease is caused by fission fungi in the feces gaining access to the extremities cannot be accepted, for the disease is not contagious, experiments with the intestinal contents not having proved effective in transmitting the disease to healthy animals. Rabe's view that the disease is really a dermatophagus scab cannot be acknowledged as correct, since the mites are frequently found on the fetlocks of healthy animals and are demonstrable in only about one-third of the cases in sick animals (Johne). Finally there is no confirmation of Bräutigam's opinion who found the same micrococcus in the slop, in the intestinal contents and in the fluid of the vesicles, and considered that this caused the inflammation of the skin, owing to its increase in the skin and in the subcutaneous tissue when the former is soiled by the feces.

While experiences in general are in favor of the harmlessness of corn and wheat slops, Schröder noticed a considerable outbreak of the disease in a large herd of cattle and also in a horse from feeding on maize



slop, and the symptoms were quite similar to those of malt eczema, with this difference, however, that they assumed a much more severe form and caused the death of 12 cows and of 13 calves.

**Predisposition.** Animals newly stabled for fattening are affected first of all, that is animals which are changed from another food to potato slop; the disease therefore is observed most often in fattening establishments in which the animals are changed frequently. With the same mode of feeding milch cows do not contract the disease or only exceptionally; the disease occurs less seldom in steers that are out in the open than in animals that are constantly kept in the barn. In some animals an actual idiosyncrasy against distiller's slop appears to be present, for only in this manner can the fact be explained that in the same year and in the same stable there are animals that become ill from a few bucketfuls of the slop, while there are others that remain healthy in spite of having ingested great amounts of the slop (Johne). The conditions of the stables have no influence in this direction.

**Symptoms.** The disease manifests itself only in a later stage of feeding with potatoes and slop, developing mostly in 2 to 3 weeks, and stands in general in direct relation to the amount of injurious food ingested, now assuming a mild and again a severe form.

In the more frequent **mild form** a vesiculous inflammation of the skin is limited to the lower parts of the limbs (Schindelka); it is, however, also accompanied by general symptoms which usually precede the trouble. With a slight rise of temperature the appetite declines, defecation is retarded, tears and saliva are copiously secreted and the gait is peculiarly stiff. On the second or third day a swelling of the feet develops, from the coronet to above the fetlock joint, which may be limited to the posterior extremities, but may also involve the anterior ones or even affect them exclusively; the skin is reddened, painful, hot; the hair appears ruffled. Small vesicles appear, and after they burst a moist red surface remains, on which the secreted serous fluid dries up, forming large crusts and scabs. The eruption may extend from the metatarsus or metacarpus to the hock or carpal joint or even beyond; but after a certain time no new crusts form, while under those present the superficial layer of epidermis becomes horny; the crusts then become loose and healing results, with scaling, in 2 to 4 weeks. At the affected places the hair falls out, but with the onset of healing it begins to grow again. In rare cases the changes do not pass beyond the erythematous stage.

In the **severe form** the skin inflammation extends over large portions of skin, especially if the mode of feeding is continued unchanged in spite of the occurrence of the disease, and the animals stand in dirty, badly ventilated barns. The trouble is



accompanied by severe general symptoms. In addition to the extremities of the limbs a similar eruption occurs on the thighs, especially on their inner surfaces; further, on the skin of the scrotum or udder, under the lower belly, on the sides of the body, on the neck, tail and in the anal region. At all these places the skin, the greater portion of which loses its hair, is thrown into close folds, raw and in spots purulent, covered with thick crusts, while at the joints deep cracks are formed from which angry looking ulcers develop. In such severe cases the extremities finally become considerably thickened, abscesses develop under the skin, at the coronet the integument may even become necrotic and a purulent inflammation of the joint may arise. Sometimes suppurating ulcers with reddish edges as large as a one-cent piece are seen at the same time in the oral cavity, especially on the edges of the upper jaw (Cadéac).

Meanwhile profuse diarrhea sets in, together with total loss of appetite and great weakness, which enfeeble the animals more, so that some lie continuously on the ground and eventually die from exhaustion or in consequence of pyemia or septicemia. With the onset of diarrhea the exudation in the skin becomes less, whereupon it becomes drier and stiffer and more closely attached to the subcutaneous tissue.

In potato rash the following symptoms have been noticed aside from the usual ones: Inflammation of the hair bulbs and general falling out of the hair in horses, inflammation of the prepuce in steers and sheep, reddening of the vulva in cows, edema at different places in the skin, and finally itching of the skin in sheep; and all these have occasionally been observed without a visible outbreak of rash (Schindelka).

**Course and Prognosis.** In the great majority of cases the disease runs a favorable course, resulting in complete cure within 2 to 4 weeks. In neglected cases, however, the animals become emaciated in consequence of the increasing inflammation of the skin and of the diarrhea which often is associated with it, and the affection may then last for several months; but even here recovery may occur if the attack is limited to the feet. Cases of death are observed only exceptionally and generally only when the inflammatory process has involved the deeper layers of the skin and if the ulceration of the integument, the purulent ichorous inflammation of the subcutaneous connective tissue or of the articulations of the feet lead to a general infection.

The prognosis is generally favorable if the necessary change of food is carried out in good time; a severe course is only to be feared under very unfavorable hygienic conditions.

**Diagnosis.** The history of the case, which is usually easily obtained, that is, feeding on potato slop or potatoes in large quantities, and likewise the acute vesicular and moist character



of the inflammation limited to, or at least beginning on, the extremities facilitate the diagnosis. In the fattening barns of potato distilleries the so-called dirt eczema and dermatophagus scab are also seen frequently, but dirt scab does not extend above the fetlock joint and is not accompanied by general symptoms, while dermatophagus scab develops much more slowly and corresponds in character to chronic eczema without vesicles and moisture. The demonstration of scab mites in the crusts does not necessarily exclude malt eczema, for the parasites may also be found on the skin in this disease; it is accordingly of importance in all cases to make a diagnosis from the clinical symptoms of the disease. Ulceration of the claws, intertrigo between the claws, further foot-and-mouth disease are limited to the interdigital spaces or the edges of the coronet; the large vesicles present in foot-and-mouth disease will not easily be mistaken for those of malt eczema. Panaritium (foot rot) may indeed form a complication of distiller's slop eczema, but since more distant portions of the skin are involved in eczema, it can readily be differentiated from panaritium due to other causes.

**Treatment.** The first step in treatment is the removal of the cause by the suspension of malt feeding, by reducing the daily rations of swill or potatoes down to the non-injurious quantity, and by the addition of raw food or crushed grain, bran or oil cake. The less potatoes or slop the animals receive the more rapidly they recover, and only when all bad symptoms have disappeared, the daily quantity of the previously injurious food may again be gradually increased; yet it appears inadvisable, at least at the fattening period, to feed the maximal quantity. If dry food is not procurable in sufficient amount, one can lessen the injurious effect of the malt by making a mash of one-third of corn and two-thirds of potatoes (Märker), besides the addition of chalk (50 to 100 gm. to 50 liters of swill) or lime water (2 to 3 liters daily) is advantageous (Haubner & Siedamgrotzky). The result of the treatment will be aided by daily exercise of the animal in the open. Eggeling saw the disease disappear from a herd after the distiller's waste was kept heated by steam at 60°. Musterle saw the disease disappear almost completely after the feed scalded with boiling wash had been given to the animals at a temperature of not less than 45° R.

In the local treatment it is necessary to keep the animals dry and to litter their stalls well with dry straw. Otherwise the principles of treatment are the same as in eczema (see page 863).

**Literature.** Baranski, Ö. Rev., 1886, 65.—Bräutigam, Inaug.-Diss., 1886 (Lit.).—Eggeling, Pr. Mt., 1881-82, 58.—Johne, S. B., 1877, 143 (Lit.).—Märker, Handb. d. Spiritusfabrikation, 1877.—Musterle, Münch. t. W., 1910, 189.—Ohlmann,



Tm. Bundsch., 1891, 70.—Rabe, Hann. Jhb., 1877-75, 80; D. Z. f. Tm., 1879, V, 284.—Schindelka, Hautkrankheiten, 1908, 380.—Schröder, W. f. Tk., 1894, 397.—Zürn, Pflanzl. Parasiten, 1889, 280.

**Other Food Rashes.** Hess frequently saw an eruption similar to that of eczema in cattle fed on malt. The extremities were usually affected and in severe cases the udder as well, and in such cases a general disturbance of health was observed. Recovery occurred in 8 to 30 days. For treatment 10 to 30% of creolin ointment was recommended. Schmidt observed in several cows a nodular eruption with subsequent formation of hemorrhagic cracks and scabs. (Hess, Kongr. Bern., 1895, 286.—Schmidt, W. f. Tk., 1903, 273.)

Large amounts of the skins of pressed grapes (Röll) or the husks of grapes and vine leaves (Faller) given as food cause an eruption similar to malt eczema which is accompanied by diarrhea. (Faller, A. f. Tk., 1899, XXV, 225.)

Reinhardt saw an eczematous eruption on the extremities, round about the eyes and mouth in 3 calves fed on rice bran (W. f. Tk., 1891, 87).

Döderlein observed in cattle an exanthema like that of malt eczema after feeding on white mustard (W. f. Tk., 1896, 77).

Sipp described an attack of dermatitis similar to slop eczema; it had occurred in cows and bullocks after feeding the residues of pressed beet-root and affected the skin of the extremities, head and neck (Pr. Mt. 1852-53, 65).

An enzootic of hop exanthema was recently reported by Zaruba in a large cattle herd. The disease occurred year after year on this estate after the animals had daily received a certain quantity of tendrils of the hop plant freed from the umbels, and after a bad harvest the disease showed a wide extension. The skin of the udder and of the posterior extremities was affected mostly; in some animals, however, the fore extremities and the lower chest, and in one case the whole surface of the body were attacked. The clinical picture agreed in general with that of malt eczema. The cessation of hop feeding, exercise in the open and suitable local treatment produced a cure within 10 days (T. Z., 1907, 577).

Röder saw a severe moist eczema limited to the fetlocks in three horses after feeding on peat molasses (S. B., 1900, 255).

Finally Mouilleron observed in horses an eruption similar to that of malt eczema, which occurred after feeding on corn glucose cakes. Fever and general symptoms, as well as motor disturbances were noted in the acute cases. The disease usually commences in the fold of the fetlock, gradually passing on to the anterior surfaces of the extremities, and spreads up to the hock or carpal joint. At times, the skin at the inner surface of the thigh, the prepuce, the neighborhood of the rectum, the eyes and the lips is also involved. At the last named places one misses the otherwise prominent moist surfaces, the skin appears very dry, stiff, cracked and painful. If the disease develops more slowly, moisture is slight at the extremities, and the exanthema is very obstinate; but in other cases it can be made to disappear in 3 to 5 weeks by a change of food and by proper local treatment (Ree, 1907, 569).

(a) **Buckwheat Rash. Fagopyrismus.**

Buckwheat exanthema is a skin disease occurring from the simultaneous effects of buckwheat and sunlight; according to the intensity of these factors it may occur either as a simple redness of the skin or, as a more or less intense inflammation, which may even lead to necrosis of the skin.

**Occurrence.** The disease is found chiefly in sheep; swine, cattle and goats being attacked much less often, and horses only exceptionally. Only white or white spotted animals are affected. The disease appears only in certain years and in certain localities (Dammann).

**Etiology.** The cause of the disease is found in the ingestion of buckwheat (*Haidekorn*; *Polygonum fagopyrum* and *P. persicaria*). The green flowering plant is most dangerous, but the grain, the straw, the chaff and the bran may also produce the disease. The eating of buckwheat fodder alone will not cause the disease, the effect of sunlight on the white or white spotted parts of the skin being also requisite.

The immediate cause of the skin affection is not yet known, and it is not even possible to offer an entirely satisfactory explanation why the combined influence of the buckwheat fodder and of the sun rays is always necessary to produce the disease and why the feeding of the same material is harmless for black animals and for the black portions of the skin. Experience has shown that both black animals and animals that were dyed black remained free from the disease, as did also the pigmented portion of the skin of spotted animals, and that white animals whose hide was covered by dust and dirt were affected less severely, other things being equal. On the other hand, it has also been observed that buckwheat fodder usually is not effective even in white animals if it is given in cloudy weather, in winter or in the barn. It is not necessary for the occurrence of the eruption that the animals should be exposed to the action of sunlight at the same time that they ingest the buckwheat fodder; cases have been observed in which the disease followed the exposure to sun rays in 3 to 4 weeks after a copious ingestion of such food. The combined action of buckwheat and sunlight was recently determined experimentally on white mice and guinea pigs by Öhmke, who found that buckwheat loses its injurious effect by extraction with alcohol and that the extract exerts a toxic effect upon white animals on exposure to sunlight.

Dammann believes that the cause of the disease is found in certain fungi in the buckwheat which come in contact with the skin and which injure the unpigmented parts of the skin, either themselves or through their poisonous products.

**Pathogenesis.** Buckwheat food evidently contains poisonous substances which may develop in the plant itself under certain conditions of soil or through the influence of microorganisms. The experiments of Öhmke do not eliminate the correctness of the view that poisonous substances form in the digestive canal after the ingestion of infected food and then are absorbed, just as are those that may be present in the fodder. According to Schindelka, the affection of the skin is produced by toxins



which are circulating in the blood and which cause an injury to the vasomotors under the influence of the chemical rays of the sun, in those portions of the skin in which the action of the sun rays is not prevented by pigmentation. The functional disturbance of the vasomotors is then said to lead to changes in the vessel walls. The same substances are also supposed to produce certain disturbances in the digestive organs and in the central nervous system.

**Symptoms.** The exanthema develops mostly on the face and on neighboring places such as the ears, the throat and possibly the neck.

In mild cases an erythematous inflammation of the skin occurs which manifests itself by intense redness, slight swelling and sensitiveness of the skin at the affected spots as well as by itching. The symptoms diminish in one or two days and the animal recovers, yet desquamation as well as brownish discoloration of the affected parts of the skin are observed for some time.

In severe cases the clinical picture of a vesiculous or erysipelatous inflammation of the skin is presented; the deeply reddened and painful skin swells considerably at the places previously mentioned, and in consequence the ears droop limply. Often lentil to pea-sized vesicles with clear contents form on the skin (so-called head- or pox-erysipelas of sheep); after they burst moist spots form, which, however, become covered with crusts on drying of the secretion. At this time violent itching exists which causes the animal to shake its head and rub it against fixed objects. Generally brain symptoms are also noticed, the animal running restlessly to and fro, leaping about wildly, executing forced movements, or appearing as if badly stunned. In many cases the animals also have convulsions. The appetite is more or less diminished.

If the brain disturbances are considerable or if dyspnea occurs as a result of narrowing of the nasal passages, a fatal result may take place after 8 to 12 hours, otherwise the disease usually results in recovery in a few days, especially if the animals are led to a shady and cool place after the onset of the first symptoms.

**Treatment.** Change of food and placing the animal in a cool and shady place or into the barn soon bring about improvement. The administration of mild purgatives (castor oil 50 to 250 gm., neutral salts 50 to 100 gm.) is indicated. If severe inflammatory symptoms are present, local treatment must be carried out consisting in cold compresses with clean water or with lead lotion, possibly also in washing with lime water, while later inunctions of carbolized oil (1:10), lime water and linseed

oil on the reddened skin, as well as a drying powder (such as powdered zinc oxide and starch equal parts) do good service.

**Literature.** Dammann, *Gesundheitspfl. d. Haustiere*, 1902, 322.—Henninger, *B. Mt.*, 1886, 15.—Klein, *A. f. Tk.*, 1891, X, 220.—Noack, *S. B.*, 1897, 141.—Öhmke, *Z. f. Physiol.*, 1909, XXII, 685.—Schindelka, *Hautkrankheiten*, 1908, 376.

### (e) Clover Disease.

Clover disease consists in a variable inflammation of the white portions of the skin of the head and of the limbs, also frequently affecting the mouth; general symptoms of illness may sometimes be noticed.

**Etiology.** Especially after generous or exclusive feeding of Swedish or bastard clover (*Trifolium hybridum*), the disease has repeatedly been observed in horses (Dammann, Haubner, Zipperlen, Michael, Heimann), Kováts saw it also after feeding on red clover (*Trifolium pratense*). Enzootic outbreaks have been noticed in cattle in consequence of pasturing in clover meadows (Berndt, Nevermann) or after feeding with red clover (Nissen). Sheep also have been thus affected (Berndt, Nevermann).

The actual cause of the disease is not yet known. According to Haubner it is due to fungi, but this view cannot be considered to be proved. One might assume that the poisonous effect of the clover is somewhat similar to that of buckwheat in fagopyrism (see page 873).

**Symptoms.** In mild cases there is merely a redness, perhaps also a moderate swelling of the white parts of the skin on head and limbs, which soon disappears and leaves behind a somewhat prolonged desquamation. In cattle the skin of the extremities, of the udder, and of the lower belly are favorite localizations of the eruption, yet the front of the chest, the lower part of the neck, and the parts around the mouth may also be affected.

In a severe attack the parts of the skin mentioned above become dark red to bluish red in color, considerable swelling and tenderness may be noted sometimes followed in places by vesicles, and then a large quantity of yellow colored gummy fluid exudes upon the skin which soon dries into rather thick scabs. Collections of pus then form, whereupon it exudes from fissures in the scabs which have in the meantime become cracked, or a purulent layer is disclosed after falling away of the scab, which generally takes place within 14 days. Itching and inflammation of the hair bulbs were observed by Haubner, and Kováts saw inflammation of the lymphatic vessels with the



formation of abscesses along their course. In case of an intense intoxication the affected portions of the skin may become subject to dry necrosis.

In severe cases in horses phlegmonous stomatitis (see page 196), jaundice, colic and nervous disturbances similar to those seen in fagopyrismus were noted in addition to the symptoms described (see page 874), also amaurosis and paralysis (Friedberger & Fröhner). In cattle Nissen very often saw symptoms of pseudo-aphthous inflammation of the mouth (see page 193) with the formation of diffuse, yellow croupous deposits on the mucous membrane of the lower lip, on the hard palate (especially between its bars), and at the posterior part of the lingual ligament. Some animals even presented the symptoms of a purulent conjunctival catarrh.

Cases accompanied by severe nervous and digestive disturbances generally lead to death in a short time, but in the other cases recovery ensues with suitable treatment.

**Treatment.** In slight cases it is sufficient to stop the clover feeding in order to secure a quick recovery of an ailing animal, while in bad cases the addition of local treatment with cold applications appears necessary; for these, clean water or better Goulard's extract or lead lotion, or Burow's solution may be employed, and afterwards the treatment may be similar to that applied in eczema (see page 863). The nervous disturbances require symptomatic treatment, while the inflammation of the mouth is to be treated on the principles already outlined elsewhere (see page 198).

**Literature.** Berndt, Pr. Vb., 1905, II, 23.—Heimann, Z. f. Vk., 1909, 490.—Jakobs, B. t. W., 1905, 790.—Kováts, Vet., 1895, 266.—Michael, S. B., 1898, 112.—Nissen, Maanedsskr., 1909, XX, 602.—Zipperlen, Rep., 1885, 163.

**Exanthema Caused by Other Leguminosæ.** Kühn and Bigoteau saw an extensive eruption of an eczematous nature in cattle after feeding on lucerne if this fodder was given in large quantities, especially if it was young and luxuriantly grown; the same observation was made by Marek. Burmeister noticed a similar skin disease in horses which had received vetches in addition to lucerne.

The symptoms in cattle are similar to those of malt eczema (see page 869), only that the eruption, which also commences on the fetlocks of the hind legs, spreads rapidly over the whole of the lower legs, and sometimes over the inner surface of the thighs, the udder or the scrotum, and even over the lower abdomen as well as the anterior extremities. The skin of the lower chest and of the breast may also be involved (authors' observation). There is swelling and redness of the skin as well as vesicle formation, whereupon copious serous fluid is secreted which dries into thick scabs which subsequently crack. Under the scabs much pus is collected. Considerable tenderness is always manifest.



In horses the disease occurs either at the same time on the skin or mucous membrane of the mouth, or only the skin of the limbs covered with white hair is attacked, leading to edematous swelling, the secretion of sticky fluid on the surface of the skin, and scab formation.

Besides stopping or reducing of lucerne feeding, the treatment is the same as that in malt or moist eczema (see pages 863 and 871).

**Literature.** Bigoteau, Bull., 1894, 456.—Burmeister, Mag., 1844, 112.—Koványi, A. L., 1907, 471.—Kühn, B. t. W., 1894, 521.

## 9. Erysipelatous Inflammation of the Skin. Dermatitis Erysipelatosa.

(*Rotlauf, Rose; Erysipelas.*)

By erysipelatous dermatitis is understood a diffuse, sero-cellular infiltration of all the layers of the skin, which is manifested by an intense red color, pain and swelling, and which after an acute course passes on to healing with desquamation of the skin.

In the strict sense the name applies only to diseases caused by Fehleisen's streptococcus erysipelatis. It is, however, unknown whether this etiologically uniform type of disease—idiopathic and wound erysipelas—also occurs in animals, but cases which have been met with actually correspond with this disease very much clinically, and in consequence some authors (Röll, Malzew, Semmer, Lucet) have considered them to be identical with true erysipelas.

From the definition given it is clear that the red spots developing in swine erysipelas do not correspond to the meaning of the pathological term of erysipelas, but have merely the significance of redness of the skin.

**Etiology.** The external influences mentioned in the etiology of erythema (see page 839), especially the effect of too great warmth, sun rays as well as chemical substances, produce on intense exposure an infiltration of the skin with sero-cellular exudate extending into the subcutaneous connective tissue. Brandes also proved experimentally that kainit mixed with the litter or strewn in the manure pile produced violent inflammation of the skin and even necrosis of the extremities and udder, and if taken into the stomach may cause a fatal internal disease, especially in hens. Traumatic effects especially play a part as means of infection. From such causes the disease develops on the head and limbs and seldom on other parts of the body, especially in horses. The same origin is probably to be attributed to the quite common erysipelatous dermatitis on the head and neck in swine (Schindelka), as well as to the inflammation which now and then occurs in dogs (Fröhner, Müller).

Lucet isolated a streptococcus quite similar to the streptococcus erysipelatis from the affected skin of a horse that had died with symptoms of erysipelas of the head. This streptococcus was demonstrated on microscopic sections in the distended lymph spaces which were filled with exudate.



Certain food rashes, especially buckwheat exanthema and clover or lucerne disease, give rise in severe cases, to symptoms similar to those of an erysipelatous inflammation of the skin (see pages 874 and 875).

Straub has noted an erysipelatous inflammation of the fetlock region in military horses after drill on stubble fields, while Späth saw a similar disease in cattle that were littered on pine and fir needles. Cadéac and Nys attribute a vesicular inflammation of the skin of horses to the action of an acrid substance of *Blaps mortisaga*. The affection occurred in the months of June and July, and especially affected the head and the region about the mouth. Kossorotow noticed in horses which had been ridden over fields thickly grown with wolf's milk (*Euphorbia*) a disease which was manifested by an inflammation of the skin, with moderate fever, redness, excoriations and rhagadæ.

**Symptoms.** Erysipelatous inflammation begins either with small spots which spread and merge into one another, or it may commence with large uniformly reddened patches, in horses for instance at the ends of the extremities; there is uniform redness of the skin, swelling, elevation of temperature and increased sensitiveness on handling. These morbid changes which may moreover be accompanied by intense itching, remain unchanged for several days, later on, however, the skin becomes softer, the brownish epidermal layer desquamates in fine or coarse scales, and underneath the healthy skin reappears. In an intense inflammation large and small vesicles develop (*Erysipelas vesiculosum et bullosum*) which soon burst or suppurate, whereupon the surface of the inflamed skin is for a time covered with crusts (*E. crustosum*). The development of dermatitis is often accompanied by a feverish rise of temperature, loss of appetite and depression, but these symptoms disappear as soon as desquamation commences.

The pathological processes show certain peculiarities, according to their cause and even more according to breed of the animal, especially as regards the localization of the changes. Thus painful hot swellings develop in horses on the head, around the orbits, the nasal openings and the cleft of the mouth, in consequence of which respiration as well as mastication may be greatly hindered, while at other times the inflammation on the unpigmented extremities occurs at the coronets, on the fetlocks and shin bones, and then considerable thickenings develop in consequence of infiltration of the subcutaneous cellular tissue which also recede after cessation of the inflammation.

A peculiar form of erysipelatous dermatitis was observed by Lebrun as an enzootic in military horses. Vertical wounds running parallel with one another developed in the region of the cleft of the mouth and became covered with crusts. At the same time there was pyalism and hyperemia of the mucous membrane.

**Diagnosis.** Erysipelatous dermatitis manifests itself by diffuse inflammatory changes in the skin and by its acute development accompanied by fever. This distinguishes *erysipelas* from the much milder *erythema*, further from *eczema* which

develops more slowly, extends only gradually and is usually afebrile. But in eczema the small vesicles develop from the beginning while the skin is still comparatively healthy, while in erysipelas large vesicles form only later when the skin is intensely inflamed. In phlegmons purulent infiltration of the subcutaneous connective tissue exists.

**Treatment.** Local treatment is only necessary in severe cases of erysipelas, while slight cases heal without any treatment. Cold applications of clear water, Goulard's extract, lead water, also Burow's solution, or applications of lime water may be used. Later on the reddened skin may be anointed with carbolyzed oil (1:10), or covered with astringent powders (see page 863). In cases where erysipelatous inflammation is caused by an infection, a subcutaneous injection of 3% carbolic acid or 1:5000 corrosive sublimate solution at several points of the periphery of the affected spot (in horses one can inject 10 to 30 cc. at one place) checks the advance of the inflammation (Gutzeit, Feldmann). The following may also be used: 10 to 20% ichthyol, resorcin, creolin, iodoform ointment, then gray mercury ointment, or disinfecting solutions. Where considerable general disturbance of health exists corresponding symptomatic treatment is indicated.

**Literature.** Cadéac, J. vét., 1902, 515.—Gutzeit, Z. f. Vk., 1892, 301.—Kossorotow, Pet. A. f. Naturwiss; 1897, 42.—Lebrun, Bull., 1906, 286.—Nys, Rec., 1907, 44 (Rev.).—Späth, Mt. d. Ver. bad Tzte., 1903, 86.

## 10. Gangrene of the Skin. Gangraena Cutis.

(*Gangrenous inflammation of the skin; dermatitis gangrænosa.*)

By gangrene of the skin is understood a death of circumscribed portions of skin, which is produced either as a direct result of tissue destruction, or from an arrested blood supply to the part, or which arises in the course of deep seated inflammations of the skin.

**Etiology.** In the unpigmented and perhaps sparsely haired skin the chemically acting (ultra-violet) rays of the sun produce hyperemia on prolonged and direct exposure, and soon after erysipelatous redness and inflammation, and if their effect on the skin is very intense, they may finally cause death of the skin in these places. This form of gangrenous inflammation of the skin (*gangraena solaris*) occurs mostly on the white spotted extremities and on the white marks on the head in horses; it is noted exceptionally in cattle. Aside from the sun rays, hot dry winds may cause the same effect, and similarly gangrene of the skin may also result in consequence of freezing (*congelatio*) or burning (*combustio*).



Chemical substances circulating in the blood may cause dry gangrene of the skin. This is observed most frequently in ergot poisoning in which parts of the skin and even whole parts of the extremities, the ears, the tail, in fowls the comb, the wattles and tip of the tongue, in ducks the beak, etc., mortify and are cast off (*Ergotismus gangrænosus*). The cutaneous gangrene occurring sometimes in clover disease is likewise caused by chemical poisons. Necrosis of the skin in horses is also sometimes a symptom occurring in lupinosis; *Lathyrus sativus*, lucerne and buckwheat may produce a like action on the uncolored parts of the skin. A similar gangrenous inflammation develops on the extremities of horses on the coronets and fetlocks, sometimes without any assignable cause (so-called necrotic eczema) in which, however, the injurious effect is probably due to bacteria.

The frequent skin gangrene occurring in swine is mostly caused by the bacilli of swine erysipelas (Jensen), which multiply in the vessels of the skin, producing in mild cases a serous infiltration (urticaria) and in severe cases a tissue necrosis. The same thing may also be noticed quite frequently in swine plague or hog cholera. In the course of purpura hemorrhagica, gangrene of the skin may occasionally be observed. Other pathogenic bacteria (*Bac. necrophorus* and perhaps others) doubtlessly play a part in cases where a quick destruction of neighboring tissues results from insignificant injuries.

In the Hungarian stud at Mezöhegyes in a stable where a slight inflammation of the heels of the animals had been noticed every year in the months of July and August, in the year 1889 gangrenous inflammation of the fold of the fetlock occurred among 1 and 2 year old stallion foals which were kept here, and assumed an enzootic character. Thirty foals became affected and the disease assumed such a severe character that in some cases gangrene of the tendons, ligaments and bones developed. The cause was probably an infection with the bacillus of necrosis.

Continuous pressure on the skin at times causes mortification in consequence of disturbing its nutrition, and this may frequently be seen in animals lying on the ground for a long time, on parts of the skin which immediately cover bones. The same is brought about by severe contusions.

Finally, corrosive substances such as concentrated acids, alkalies, etc., cause a mortification of the parts that are touched immediately as well as neighboring portions of skin, in consequence of coagulation and destruction of the cell albumen.

**Symptoms.** Gangrene of the skin is preceded either by acute eczema, in which case reddening, increased sensitiveness and swelling of the skin are noticed, or it presents itself immediately without such prodromal symptoms. After the onset of necrosis the skin becomes brown, and at the same time its



sensitiveness diminishes to complete anesthesia. If the process remains more superficial, only the epidermis of the stiff and dry skin dies and is thrown off in thick lamellæ from the cutis, which is then covered by a fresh outer skin.

If, however, the necrotic process extends more deeply, the skin in its whole thickness turns into a black, dry, parchment-like layer, round about which acute inflammation may often be noticed. The pus exuding from this inflamed wall separates the dead portion of skin, which is finally cast off, and the ulcerated surface remaining in its place heals by granulation and cicatrization, unless a general infection is associated with the inflammation. In more severe cases the necrotic inflammation extends still deeper, to the tendons and ligaments, whereupon a purulent ichorous process arises in the neighboring joints, which leads to destruction of the joint surfaces and finally to general infection by metastasis. This course is especially to be feared on the extremities of horses, cattle and sheep, and even if no fatal pyemia or septicemia intervenes, the animal becomes quite useless because the joints become stiff or the cicatrices contract very much, thus interfering with motion.

In many cases of wound infection the inflamed skin changes to a greasy, pulpy, dirty colored mass (*Gangræna humida*). The process can be arrested after a time even in these cases, and after throwing off of the necrotic tissue, a healthy granulation tissue develops, followed by cicatrization, while in other cases a general infection is associated at times with the local complaint.

In dry necrosis (*mummificatio*) the skin alone or together with its subjacent layers becomes insensitive without exhibiting any inflammatory symptoms; the skin shows a black, dry appearance, while under it the tissue is hemorrhagically infiltrated. Finally the dead portion is cast off from the living tissue by sloughing.

**Treatment.** In commencing gangrene the friction of the affected parts of the skin with desiccating (lead, zinc oxide) or disinfecting (iodoform, carbolic, boracic) ointments, seems indicated. The dead parts should be removed with the knife and the remaining ulcerous surfaces treated according to the rules of surgery. Sloughing is hastened through the production of hyperemia by means of warm fomentations and poultices.

**Literature.** Cuillé, *Rev. gén.*, 1905, VI, 457.—Fröhner, *Monh.*, 1901, XII, 205.—Jensen, *D. Z. f. Tm.*, 1892, XVIII, 40, 272.—Paszotta, *Monh.*, 1901, XII, 256.—Sequens, *Vet.*, 1896, 471.—Tátray, *ibid.*, 1895, 161.

**Necrobacillosis of Sheep.** Skin necroses caused by the bacillus *necrophorus* occur especially in sheep, and not infrequently in enzootic extension. The respective cases of disease have been designated variously, according to the localization of the lesions. (Concerning the



relations of necrobacillosis to aphthous inflammation of the mouth in sucklings, see page 188.)

To this disease belongs especially the so-called sore mouth of sheep (*Impetigo labialis*), which generally attacks only lambs and commences at the edges of the lips with small vesicles, which are filled with clear fluid. At the same time the lips appear more or less swollen, and in severe cases the appetite is diminished. By rubbing of the mouth, and sometimes in taking food the vesicles burst and leave intensely reddened, bleeding surfaces which afterwards become covered with brown crusts. By the formation of fresh vesicles in the neighborhood the process spreads to the angles of the mouth and to the region of the nose, and the skin of the lips is transformed into a raw, granulating surface coated with dirty masses which appear either purple red or yellowish white and are always covered with broad cracked scabs. If the process is fairly extensive, a purulent mass makes its appearance on pressure upon the crust. On the inner surface of the lips one finds only exceptionally raw granulating surfaces which are more frequent on the gums and hard palate, and a peculiar smell, reminding one of Limburger cheese, is emitted from the mouth. The nutrition of the animal becomes poor in consequence of the painful complaint and many animals even die.

In certain outbreaks the disease attacks the neighborhood of the nasal openings, the cheeks and the eyelids, or in some animals it may occur on the extremities where similar changes develop as on the lips. This form of the disease is designated by Mohler as "lip and leg ulceration of sheep."

From its localizations on the coronary band the disease is also known as "foot scab of sheep" ("foot rot of sheep" in America, "contagious foot rot" in England, "piétin contagieux" in France). The inflammation begins at the coronary band and small ulcers develop at the heels, which discharge a purulent secretion, emitting a peculiar odor. In severe cases the process extends more deeply and fistulæ may form, sometimes also necrosis of ligaments, tendons and bones (Mohler & Washburn).

Owing to its localization on the genital organs the disease manifests itself in female animals by a painful swelling of the vulva, by ulceration on the vulva and on the adjoining skin, and by a slimy or purulent discharge from the vagina. In rams and not infrequently in wethers, the disease begins with the formation of pale yellow small spots in the skin of the prepuce, the preputial opening and on the penis. These spots soon change into ulcers, which gradually become larger, often coalescing with one another, so that the whole surface of the sheath becomes ulcerous. The sheath is generally more or less swollen and reddened.

The various forms of necrobacillosis just described now and again occur combined with one another. Thus contagious foot rot or the disease of the genital organs may be observed together with *impetigo labialis*; indeed all three forms of the disease can be present at the same time.

Necrobacillosis of sheep has been found principally in North America, England and France as an enzootic (MacFadyean, Berry, Williams, Knowles, Flook, Moussu, Besnoit), but outbreaks have also been recorded in Germany (Peter, Hasenkamp, Pr. Vb.), in Hungary (Vigadi) and in New Zealand (Giltruth).

While the bacillus of necrosis is the actual cause of the changes, predisposing factors play an important part in the spread of the disease, rendering possible the entrance of the bacilli into the tissues of



the body and a rapid extension of the disease in a flock. Of special importance in this connection are abrasions or wounds of the mucous membrane or of the skin. Such lesions easily occur on the lips and the edges of the nose from the ingestion of very rough or hard food, or when feeding on pastures in which thorny, prickly shrubs are accessible. Consequently numerous cases are noticed, especially in dry years. Abrasions and wounds easily arise on the extremities by travelling over recently graveled roads, on frozen snow, and also by pasturing on meadows containing thorny, prickly plants. The skin of the vagina and that of the prepuce is easily subject to abrasions in the covering act. As further predisposing causes may be noted: a diminution of the power of resistance of the skin through cold, standing in dirty, soiled straw and travelling on muddy roads. Weakness and insufficient nutrition also cause a predisposition to the disease.

At times, however, necrobacillosis seemingly appears without the intervention of any predisposing factors, attacking especially the mucous membranes, principally in very young animals, owing to the softness of their tissues or probably to an increased virulence of the bacillus.

Artificial transmission of the disease was successful by rubbing the crusts into the scarified skin (Mead), on the mucous membrane (Vigadi), by putting a pledget of cotton impregnated with the discharge from the genital organs into the preputial opening (MacFadyean) and by inoculation of cultures of the necrosis bacillus. In an enzootic in the Prussian district of Wittenberg, the shepherd was also infected by the affected sheep, and large lentil-sized nodules formed on his hands, which in 3 to 4 days changed into small ulcers and healed up in about 10 days.

The course of the disease is generally benign, yet now and then losses of 10% of the affected animals have been noted.

The treatment consists in removing the crusts and granulations and then in washing or swabbing the places with disinfecting fluids. When the disease is localized on the lips and edges of the nose, inunctions with a 5% cresol or tar ointment, to which 10% flowers of sulphur may be added are more appropriate. The sheath or vagina may be syringed out daily with a 2% solution of potassium permanganate or with 75% hydrogen peroxide. Animals with extensive ulcerating surfaces or strongly granulating ulcers are best killed.

Preventive measures are cleanliness and the disinfection of the quarters of the animals, isolation of new bought animals for a period of two weeks, isolation of diseased subjects and the avoidance of infected pastures for the space of a year.

**Literature.** Kondor, Ung. Vb., 1901.—MacFadyean, J. of comp. Path., 1903, 375.—Mead, Am. v. Rev., 1905, 441.—Mohler, Lip-and-leg ulceration of sheep. Anim. Ind. Circ. 160, 1910.—Otto, S. B., 1905, 237.—Peter, B. t. W., 1899, 168.—Pr. Vb., 1901, II, 25.—Williams, J. of comp. Path., 1904, 64.

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**Necrobacillosis of Other Animals.** Besides occurring in sheep, necrobacillosis not infrequently affects other species of animals, partly as a sporadic and partly as an enzootic disease. Of these is to be mentioned first of all diphtheria of calves (see Vol. I). In Paraguay, Elmassian & Ulizar saw an enzootic of necrobacillosis in adult cattle, where the necrosis attacked the skin almost regularly in the perineal region, and only exceptionally on the tail, udder and ears.



The interdigital panaritium and the necrotic (malignant) inflammation of the claws in cattle also belong to this category (see Vol. I). Finally many cases of pseudo-aphthous inflammation of the mouth (see page 193) arise from an infection with the necrosis bacillus. This was the case in an enzootic in cattle described by Vigadi. The disease commenced here with apathy, diminished appetite and slight fever. After 1 to 2 days salivation occurred, and on the reddened mucous membrane of the mouth, especially on the inner surface of the lower lip, of the dental pad of the upper jaw and the tip of the tongue, necrotic patches appeared which varied in size from that of a lentil to that of a quarter; they were roundish, grayish white; after their removal the tissue of the mucous membrane was intensely reddened and bleeding. In several cases the necrotic process also attacked the deeper layers, whereupon deep ulcers with dirty grayish white smeary deposits were formed. Simultaneously with these changes the lips, the alæ of the nostrils and the region of the cheeks showed painful swellings, in some cases diffuse, yellowish brown scabs under which the skin was intensely reddened, denuded of epithelium, and moist. Not infrequently the skin of the feet was also affected, while the swollen and painful skin of the interdigital clefts, more frequently, however, that at the edges of the coronet, became necrotic at places varying from a one-cent to a twenty-five-cent piece in size, and gave place to bleeding ulcers with ragged edges. Now and then also grayish brown, firmly adherent scabs were formed at the same time on the swollen skin of the fetlock region which covered a moist surface denuded of epithelium. Recovery followed in slight cases within 10 days, and it occurred even in the severe cases by keeping the affected parts clean but without other special treatment.

Finally the enzootic necrosis of the vagina of cattle is caused by the necrosis bacillus, and occurs together with necrotic inflammation of the claws (Ellinger).

In goats, enzootics of inflammation of the mouth have been observed in France and Germany, the clinical appearance of which coincides with that seen by Vigadi in cattle; only that the process remained limited to the mucous membrane of the mouth, and the neighborhood of the mouth. According to Cadéac and Mohler these cases also belong to necrobacillosis.

As a further form of the disease may be noted the necrotic inflammation of the fold of the fetlock in horses, which may occur as an enzootic, and also the necrobacillosis of rabbits.

**Literature.** Elmassian & Ulizar, A. P., 1906, 969.—Pr. Vb., 1903, II, 18.—Vigadi, A. L., 1906, 423.

## 11. Herpes Labialis.

By herpes one designates a disease of the skin with acute onset which heals in a short time, and which is characterized by vesicles filled with serous fluid arising on a circumscribed surface. Such an eruption usually develops on the lips and alæ of the nostrils of horses in the course of gastro-intestinal catarrh, or in acute infectious diseases. The cause is unknown; but a local irritation of certain nerves is suspected, such as single branches of the trigeminus. At the aforementioned places thickly clustered lentil-sized vesicles develop filled with a clear yellow serum, there is also slight itching, moderate redness and swelling. After

1 to 3 days the vesicles dry up to brown crusts, and after these fall off the redness of the skin soon disappears.

Treatment is only necessary in the presence of excoriations and then an ordinary dusting powder or painting with lead acetate solution may be resorted to.

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**Herpes Zoster.** (Shingles; Zona, Fr.) In human medicine this term is applied to a peculiar vesicular eruption in which small vesicles occur profusely in the territory supplied by a definite nerve, and which may be traced to a disease of the intervertebral ganglia or the nerves themselves, or to an affection of the trophic nerve fibers. A similar affection is said to have been observed by Mégnin in a horse on whose body thickly clustered vesicles formed, which crossed the direction of the hairs, forming stripes 1 to 2 cm. broad which were separated from one another by healthy stripes of skin of like width, so that the appearance was like that of a zebra skin. Further, Hébrant claims to have seen shingles in a two-year-old dog. On one flank of this dog, a patch about 3 cm. broad was covered by sero-fibrinous masses, round about which and as far as the median line, little nodes and dried up exudate could be seen; severe itching occurred in paroxysms. After 10 days a similar eruption appeared on the opposite side of the body. At first moderate atypical fever was present. The animal recovered in 2½ weeks. A certain similarity of the complaint to moist eczema of the dog undoubtedly existed. (Hébrant, Ann., 1905, 12).

## 12. Pemphigus.

(*Blasenausschlag; Dermatitis bullosa.*)

Pemphigus is an independent disease of the skin characterized by large vesicles; in its acute form it passes on to recovery in a few weeks, in its chronic form on the contrary—and in the narrower sense of the word only this is designated as true pemphigus—vesicular outbreaks recur several times, which finally, however, end in recovery (*Pemphigus vulgaris*, *P. chronicus benignus*), or the healthy epidermis fails to form again on the constantly increasing diseased areas (*P. chronicus malignus*, *P. foliaceus*).

**Occurrence.** In animals, especially in horses (Demoussy, Dieckerhoff, Graffunder) and in cattle (Loiset, Lucet), exceptionally also in swine (Winkler) vesicular skin eruptions were observed repeatedly, which always terminated in complete recovery in a comparatively brief space of time, and which for this reason may be taken to correspond with pemphigus acutus of man, although it does not appear impossible that many cases included herein have really been attacks of urticaria bullosa or eczema bullosum. True pemphigus is undoubtedly an exceedingly rare disease in animals.

**Etiology.** Of the causes of the disease nothing certain is known. Its frequent occurrence occasionally appears to point to an infection (Loiset saw it in cattle in the form of an enzootic). Bacteriological investigations hitherto conducted have led to no positive result (Ballart saw one case of transmission of the disease from a cow, Dasch from a dog to man).



**Symptoms.** At times feverish symptoms, perhaps also digestive disturbances, herald an approaching attack, while in other cases the eruption appears without any such signs. On the skin, especially on the trunk, on the lower belly, on the inner surfaces of the thighs and elsewhere, vesicles arise upon the skin which is swollen, accompanied by intense itching or without itching. These vesicles form within 1 to 2 days and may become larger than a goose egg; they are semi-spherical or flattened and may exhibit a plate-like depression in their center. The content is clear watery or yellowish, later on it may become milky, and flows out after the thin wall bursts, while this latter remains adherent to the skin for a long time, or it is removed by rubbing, and then an intensely red surface becomes visible, which for a time continues to exude a serous secretion, but in a few days is covered with new epidermis. In this way the process heals completely in the course of 2 to 4 weeks; in rare cases, however, healing occurs only with the formation of scabs, and then fine glistening cicatrices remain at the places of the vesicles.

**Treatment.** After rupture of the vesicles occurs, it is sufficient to apply a non irritating dusting powder to the skin or an astringent ointment (see page 863); in case of very extensive eruption washing with soap and thorough cleanliness are to be recommended.

**Literature.** Basch, T. Z., 1908, 266.—Graffunder, B. t. W., 1890, 153.—Jakobsen, Maanedsskr., 1893, IV, 311.—Lucet, Rec., 1894, 244.—Paulieki, Mag., 1872, 29.—Sauer, W. F. Tk., 1902, 231.—Winkler, *ibid.*, 1891, 47.

**Pemphigus Chronicus.** Fröhner saw the whole of the body of a dog, with the exception of the head and limbs, covered with large vesicles, which after bursting, left clear bright red surfaces, with a cherry red center. These showed no disposition to heal and were continually covered with fresh crusts, until finally the animal died, completely exhausted, with symptoms of hemorrhagic inflammation of the rectum.

Lafosse saw a skin disease similar to pemphigus foliaceus, in a mule. After all the hair had fallen out, broad epidermal scales formed on the bald skin, after detachment of which the fresh layer of epidermis was soon lifted up by the exuded serous fluid underneath, and in this way new scales always were formed. The animal meanwhile became emaciated, and later on was attacked by diarrhea. (Fröhner, *Monh.*, 1892, III, 497.)

### 13. Impetigo.

(*Ecthyma.*)

By impetigo or ecthyma is understood in human medicine an exanthema chiefly affecting children, in the course of which pustules form on different parts of the body, but especially on the face; they develop on a red base without itching, and are followed later on by thick, soft, honey-like crusts. This skin affection is sometimes decidedly in-

fectious (Impetigo contagiosa) and is accompanied by acute swelling of the neighboring lymph glands; it always runs a favorable course.

According to Burke the disease develops in horses with fine skin under the influence of irritating agencies and unfavorable hygienic conditions. The resultant pustules are superficial, at first white, like mother-of-pearl, later purulent, then rupture quickly, and their contents dry up into yellow crusts; when these fall off a hairless spot remains which heals without scaling. In contrast to eczema the exanthema runs a rapid course and heals quickly; there is no itching and transmission to other animals is easy (Vet. Journ. 1890, 77).

According to Bénion a similar disease develops in young swine in the neighborhood of the eyes, more rarely on other parts of the body, with the formation of small pustules, in the place of which soft crusts arise later on. The process is accompanied by a catarrh of the conjunctivæ, nose and mouth, and heals within 2 to 3 weeks (Diet., 1888, XVI, 268).

Schindelka several times saw an eruption in old nursing or pregnant bitches which occurred independently of distemper.

As a secondary affection impetigo develops in the course of distemper, strangles, swine plague and hog cholera as well as in rinderpest.

The treatment consists in softening of the scabs or washing with creolin or soap solution and subsequent dressing with a disinfecting or astringent ointment.

**Dermatitis Lichenoides.** By this name Dagès described a disease of the horse in the course of which large, dry, bald, non-itching surfaces formed on the root of the tail, on the back, neck and thighs. After the lapse of six months the skin on these spots became painful, inflamed and infiltrated, and hazelnut-sized nodules developed on it; at the same time there was violent itching. Later on the nodules began to bleed, and thick, yellowish crusts formed, underneath which the skin was covered with pus. Finally the nodules became smaller, and in their places pea-sized elevations remained, on the surfaces of which the epidermis was thick and rough. The acute attack lasted a month, it recurred annually in winter, and the affection finally became so extensive that the horse had to be killed at the end of three years. Neither in the epidermis, nor the crusts or in the pus could vegetable or animal parasites be demonstrated, and transmission experiments on guinea pigs were unsuccessful. (Dagès, Bull., 1894, 442.)

#### 14. Acne.

(*Heat rash, summer rash, summer scab, sweating eczema of the saddle region, saddle scab, heat nodules, heat pox, nodular or tubercle rash in horses; Acne simplex s. vulgaris; Boutons* [French]; *Akne* [German].)

By acne one understands an inflammation of the sebaceous glands and hair follicles, not infrequently passing on to pus formation, and characterized by pin-point to bean-sized nodules appearing in the otherwise healthy skin.

According to Schindelka the skin diseases mentioned in the title are considered as acne since they do not exhibit the characteristics of eczema with which most authors have classified them (so-called papulo-vesicular eczema).

**Etiology.** In certain cases acne probably results from an infection with pus bacteria (Frick) which evidently enter



through the orifices of the hair follicles or sebaceous glands, or are rubbed into them. According to Frick mechanical influences only play the part of accidental causes in these cases.

The disease occurs in those parts of the body which are frequently exposed to mechanical influences, especially to rubbing, or which perspire readily. Such places are, in horses, on the back and on the sides of the chest (friction by the saddle, girth, traces) on the front of the chest and in the shoulder region (rubbing by the collar and breast strap), in the croup and tail region (friction by the breeching and crupper), as well as on the head (mechanical friction from the halter). In dogs the forehead, the cheeks, the bridge of the nose and the external surfaces of the limbs are favorite spots for acne, because these parts are preferably rubbed by the muzzle or come in contact with the hard ground.

The disease, which was studied closely by Bartke, Qualitz, Steffens, Grammlich, v. Hennings, Kalkoff, and Kupfer under the name of sweat eczema (heat pox) of riding horses, is an acne-like affection of the skin, especially prevalent in the military horses of the Prussian Army since the introduction of the new army saddle (according to Kupfer 50 horses on an average in each regiment were affected in one summer), but it is also observed when other saddles are used. The connection between the frequency of this trouble and the introduction of the new army saddle, is found in the fact that the trees of the new saddle are longer, and the wallet is put further back, extending to the sensitive lumbar region, which on motion, and especially in walking, makes decided transverse motions and furnishes much opportunity for friction between the skin and the wallet. Saddles which do not fit well to the trees and which are weighted too heavily, too much stirrup riding, lack of cleanliness in the saddle region are also of importance. The chief effect is due to friction, for the trouble does not occur under the saddle itself, but at the back of the pannel and flap. The causal action of sweat and dust is shown by the fact that the disease is observed almost exclusively in summer. Horses with bad conformation and weak tottering gait are apparently especially inclined to the disease.

At times an extensive outbreak of acne occurs in horses over the whole body in the warm summer weather (so-called summer rash) when the animals perspire profusely at work.

The checking of glandular secretion by obstruction, by dirt, of the orifices of the sebaceous glands or of the opening of the hair follicles, possibly also by medicines rubbed on the skin may, according to Veiel, occasion acne in such a manner that the glandular tissue becomes mechanically irritated as a result of the dried secretion. It cannot be denied, however, that the bacteria which are usually present on the skin, or that products of disintegration formed under the influence of the bacteria, play an active part in the causation of acne.

Whether acne which occurs occasionally after the application of certain medicaments such as tar, petroleum, paraffine, vaseline, also after continued employment of ereolin or lysol solution in wound treatment, is to be taken as due to the



closing of the glandular orifice or whether the chemical irritation produced by these substances plays a part, cannot be decided from the results of observations that have been made hitherto.

Acne arises secondarily in acariasis and rarely in the course of strangles in horses, in distemper (personal observation) and according to Fröhner in bromism.

**Predisposition.** Horses and dogs are most inclined to the affection, hogs and sheep only exceptionally. Young animals and short-haired dogs appear most frequently to be attacked by acne (Schindelka).

**Symptoms.** In horses millet to pea-sized nodules develop on the aforementioned parts of the body (Fig. 126), which are distributed either diffusely (*Acne disseminata*) or thickly



Fig. 126. Acne in the horse.

clustered together and concentrated in groups of nodules (*A. confluens*). Little vesicles form in the center, the contents of which are first clear and soon after become turbid. Soon after bursting of the vesicles the content dries to a small scab, and mats the enclosed hair. Later on the scabs fall off with the matted hair, whereupon little hairless spots remain for a long time. In some nodules suppuration occurs, and on pressure bloody pus or a thick tallow-like mass may be pressed out from them. After this the swelling disappears in a short time, and the remaining saucer-shaped loss of substance soon heals, leaving a light but rather thin-skinned bald place behind (so-called shell nodule). Many nodules retrogress without previous vesicle formation or suppuration and disappear, leaving no trace behind them.

The apparently sound skin in the immediate neighborhood is slightly swollen, only if the disease is rather severe, it becomes warmer, sensitive to pressure and harder, and the nodules themselves become much harder (*Acne indurata*). Slight itching is generally noticed in the stage of development and healing; but there is more tenderness than itching.

On the hairless or sparsely haired parts of the skin of the horse (sheath, inner surfaces of the thighs and fore extremities, lower belly, chest) Schindelka saw a peculiar folliculitis which commenced in small areas with the appearance of millet sized or somewhat larger nodules. The nodules are at first palpable, deep in the skin, later on they become prominent on the surface of the skin, some of them



Fig. 127. Acne nodes on the bridge of the nose of a dog. (After Schindelka.)

change to pustules in 3 to 4 weeks, and after they burst, roundish ulcers develop with elevated edges. These heal gradually, leaving behind cicatrices, round about which fresh nodules are formed. Some nodules, however, disappear after several weeks without previous suppuration. In exceptional cases acute swelling of the lymph vessels and lymph glands occurs. The disease may last for months. The development of folliculitis was always preceded by influenza. A similar disease was seen by Marek on the anterior surface of the forearm of a horse which had not previously been attacked with influenza.

In dogs acne is principally characterized by the prominence of inflammatory symptoms, especially if the eruption is on the bridge of the nose (Fig. 127) or on the face, in consequence of



which the swelling of the skin and its tenderness are far greater. This circumstance is perhaps explained by the fact that in the skin of the dog the hair follicles are arranged group-wise round a common duct, and that on this account several hair follicles and sebaceous glands are always affected at the same time. Suppuration of the hair follicles or sebaceous glands is noticed much oftener in dogs.

In **sheep** and **swine** the clinical form of the affection appears to be similar to acne of the horse.

**Course.** In many cases, for example in the horse, the acne nodules, usually after previous vesicle formation, disappear in 1 to 3 weeks without leaving any trace, or suppuration takes place and little bald spots are presented to one's observation. Since in both cases fresh nodules are often formed, in the meantime, in the surrounding area, the course of the affection becomes prolonged and even chronic. If suppuration takes place the perifollicular connective tissue becomes involved in the process, and then little abscesses arise which extend into the subcutaneous connective tissue, coming in contact and frequently communicating with each other. Finally, however, cure results here also by scar formation. Sometimes these abscesses do not break, and pea to pigeon egg-sized hard nodules may then persist in the skin for a long time. Finally the process may lead to the formation of so-called tallow cysts (*molluscum atheromatousum*, *Schindelka*) which arise from the cyst-like dilatation of the sebaceous glands, the walls of which are much thickened and are filled with a fatty rancid mass. They form likewise hard swellings in the skin and may attain the size of a pigeon's egg.

**Treatment.** The causes of the disease so far as they may be discovered should be removed, and if possible the horses should be kept from work; under such conditions cure frequently results in horses without any further treatment. At first gentle washing with disinfecting solutions seems to be useful, while the employment of tar preparations is contraindicated; the internal use of ichthyol with water or Fowler's solution (ichthyol, dist. water or Fowler's solution in equal parts; 5 to 20 gm. for the horse, 10 to 20 drops for the dog) may do good service (*Schindelka*). Salicylic ointment is used to advantage in the incipient stage (5:100) (*Müller*). Later on the dilated nodules are pressed out, perhaps after previous splitting with a pointed knife or opening with Paquelin's thermo-cautery (*Frick*), and the resulting wound and its neighborhood is carefully washed and dressed with any disinfecting fluid (tincture of iodine 5%, pyoctanine solution 1:1,000, corrosive sublimate, etc.).

The before mentioned folliculitis of the non-hairy parts of the body in horses was influenced by treatment only insofar as the tenderness was lessened after washing with Burrow's solution, and no fresh crops of vesicles occurred after



the internal employment of Fowler's solution. On the other hand the administration of sulphur, ichthyol, resorcin, and salicylic acid preparations always resulted in the abundant occurrence of fresh nodules.

In the so-called sweating eczema of the saddle region, the following remedies have been applied: Priesnitz's compresses with creolin or Burow's solution and subsequent inunctions of vaseline, lead or boracic ointment (Grammlich), sponging with corrosive sublimate or creosote solution (hydrarg. bichlor. corros. 2 parts; creosote 5 parts; glycerine and alcohol of each 100 parts [Mauke]), finally the application of a liniment consisting of 100 parts of alcohol and two parts each of bacillol, soft soap and Peruvian balsam (Wilde). In recent times the disease has been treated successfully by cleansing the parts with softening disinfecting solutions and using drying and protecting substances on the diseased places. Kurze produced the best results by fomentations with warm infusion of hay seeds.

As a prophylactic measure it is well to pad the harness or muzzle and always to keep them clean. Against the occurrence of so-called saddle scab, Kupfer uses oilcloth or Victoria-Battist behind the edge of the panel and flap.

**Literature.** Bartke, D. t. W., 1897, 214.—Frick, *ibid.*, 1898, 365.—Fröhner, *Monh.*, 1890, XI, 410; 1903, XIV, 461.—Gräbenteich, *Z. f. Vk.*, 1907, 323.—Grammlich, *ibid.*, 1899, 262.—v. Hennings, *ibid.*, 1900, 75.—Kalkoff, *ibid.*, 1901, 140.—Kupfer, *ibid.*, 1905, 27.—Mauke, S. B., 1906, 186.—Mrowka, *Z. f. Vk.*, 1905, 493.—Müller, *Vortr. f. Tierärzte*, 1890, 2, H. 16.—Schindelka, *Hautkrkh.*, 1909, 359.—Steffens, *Z. f. Vk.*, 1896, 163.

**Comedo.** (Grubs in the skin, *acne punctata*.) Comedones are cylindrical plugs secreted in the sebaceous glands, which often appear colored black at their outer end and may be pressed out as whitish or yellowish worm-shaped bodies. According to Schindelka, they occur quite frequently in domesticated animals, but are most frequent in dogs and swine on the hairless or sparsely haired parts of the body. The plugs are expelled in time or inflammation or suppuration of the sebaceous gland (*acne*) may occur.

**Furunculosis.** Furuncle arises through the extension of *acne* inflammation from the walls of the sebaceous glands to the neighboring tissue with subsequent death of the hair bulb and the immediately adjoining tissue. The tendency to frequent affection with multiple furuncles, which is occasionally met with, especially in dogs, is designated as furunculosis.

The symptoms of furuncle are similar to those of *acne*, except that the inflammatory focus is much larger, the pain more intense; in the surrounding tissue an inflammatory edema is present, the neighboring lymph glands are often enlarged, and within the focus one finds the dead piece of tissue in the center of the pus. In these cases general septic or pyemic infection may follow (Frick). The persistence of the disease in dogs through the formation of abscesses and ulcers requires operative measures, which consist in timely splitting of the abscesses and removal of the pus together with the necrotic tissue (Fröhner, *Monh.*, 1890, I, 410).

A furunculosis-like disease of sheep was noted in the year 1905 by Teetz in Germany in two flocks of ewes and lambs. With general symptoms of weakness there was intense swelling of the lips and the skin of the angle of the jaw down

to the chest, the wool fell out in this region, and closely placed openings formed upon the gray-blue skin which were almost circular, pea-sized, with yellowish, brittle, plug-like contents and a bad odor. Washing with creolin water, inunctions with creolin tar ointment and deep splitting as well as curetting of the dead parts caused recovery with only one death (Teetz, B. t. W., 1905, 791).

Zschokke saw a number of nut-sized, bluish red furuncles on the back of a pig affected with cuticular anthrax.

**Sycosis.** By this is understood an inflammation of the follicles of the long hair, whereby nodules or pustules form which are of variable size; from their center a hair shaft protrudes. The causes are mostly the same as in acne. The disease has hitherto been noted on the parts of the skin covered with long hair, in horses as well as in dogs, and requires treatment similar to that of an acne rash.

### 15. Contagious Pustular Inflammation of the Skin. Dermatitis Pustulosa Contagiosa.

(Socalled *English* or *Canadian* [American] *horsepox*; *Dermatitis pustulosa canadensis* [AXE], *Acne contagiosa* [DIECKERHOFF].)

Contagious pustular dermatitis is a skin disease peculiar to the horse, in the course of which pustules arise on the swollen skin which appear chiefly at those places which come in contact with the harness and which may be as large as lentils. The pustules are caused by the acne bacillus of Dieckerhoff & Grawitz, which in its turn belongs to the group of the bac. pseudotuberculosis of Preisz.

**History.** The disease was first described by Goux (1841) and at the same time was recognized as a contagious disease. Bassi saw it in the year 1876 in Italy, in English and American horses, Axe in the year 1879 in England; by this author it was called dermatitis pustulosa canadensis, since in his opinion the affection was brought to England by Canadian horses. Since then the disease has been observed frequently in Europe and its etiology has recently been investigated. Thus Schindelka considers that it is identical with the impetigo contagiosa of man and he found micrococci in the pus as causes of the inflammation; Siedamgrotzky succeeded in transmitting it to rabbits and guinea pigs by inoculation. By bacteriological and inoculation experiments Grawitz & Dieckerhoff proved a bacillus to be the cause of the disease, the classification of which was more exactly established by Nocard.

**Occurrence.** The disease occurs as an enzootic, especially in horses of the English breed.

**Etiology.** The fission fungus described by Grawitz & Dieckerhoff as the acne bacillus is about two microns long; it multiplies by division, forming coccus-like chains which remain connected with each other for a time in 2 to 4-linked series. The bacilli may be stained with the usual aniline dyes and also by Gram's method.



**Cultivation.** The bacilli grow best at body temperature on solidified cattle or horse blood serum; they assume the form of small round colonies which are at first pure white and later on yellowish gray.

**Pathogenicity.** The introduction of a few drops of the culture diluted with water, by friction, into the healthy or superficially scarified skin of a horse produces typical pustules. In calves, dogs and sheep the effect is similar but milder. In the rabbit, on the contrary, a severe inflammation of the subcutaneous connective tissue develops which may lead to death, while guinea pigs die of septicemia two days after rubbing in of even a moderate amount of the culture.

**Natural infection** takes place through parts of the harness as well as through cleaning utensils. The favorite localization of the disease is on the saddle region and on the chest wall, in all probability owing to the fact that these parts are pressed on by the saddle or girths; the hyperemia produced by the pressure, and possibly also superficial losses in the continuity of the epidermis cause the infection to be established more easily in these places, but the eruption occurs also, less often, in other places, particularly on the extremities.

**Symptoms.** Two to three days after artificial, somewhat later (according to Schindelka 6 to 8, and even 14 days, at times as early as 24 hours [Lühns]) after natural infection the skin swells at one or several round or oval places from a one cent to a twenty-five-cent piece in size, becomes warmer and more sensitive, its surface moist, while the hair appears ruffled. Soon hemp-seed to lentil-sized vesicles with thin walls develop in variable number on the swollen parts of the skin; their contents, which are at first generally turbid, in a short time become purulent. The thin wall of the vesicle or pustule generally bursts after one to two days, whereupon its contents dry up and form thick, honey-yellow, gluey, tenacious, flat or centrally depressed crusts, under which grayish white or grayish green pus collects. Meanwhile a fresh layer of epidermis forms under the crusts, the crusts loosen and fall off, together with the hair, after about a week, and in their places round, hairless, colorless, non-scaling patches remain, which later are covered by new hair. During the whole time the skin between the inflamed surfaces remains healthy.

The development of the exanthema occurs without itching, without fever or any signs of ill health, only the parotid glands and the thyroids swell acutely in isolated cases, but this symptom disappears when the vesicles dry up. In mild cases the affection passes off in 3 to 4 weeks, while eruptions may arise in the neighborhood from the broken up morbid products of the affection.

After a very severe infection or by keeping animals at



work in spite of their illness, the process assumes a very severe character, the inflammation penetrating into the deeper layers; the corium dies in places and, after the falling away or removal of the dollar-sized scabs, crater-shaped ulcers make their appearance, which gradually are filled with granulations and finally heal, leaving scars. Moreover, the lymph vessels leading from the inflamed regions swell into hard, sensitive cords, in the immediate neighborhood an edematous infiltration develops, and the regional lymph glands swell or, exceptionally, suppurate. Friedberger saw the disease occur on the legs in connection with an inflammatory swelling of the joints. Finally healing takes place, but in such cases the disease lasts several weeks, and may continue as long as two months.

**Diagnosis.** The peculiar localization of the morbid processes, the pustules forming in groups on the swollen parts of the skin, the absence of itching and the contagious character of the affection, which can in all cases be proved by experimental inoculation, are distinctive features of the disease, and distinguish it from the intense itching eczema, further from acne, and also from the galls caused by saddle and harness, which occur without vesicle formation. At times the disease may arouse suspicions of farcy if the affection exceptionally develops on the extremities, if edematous swelling occurs in the region of the exanthema, and if further also inflammation of the lymph vessels and ulceration takes place; the presence of pustules and the tendency to healing, and on the other hand microscopical examination or experimental inoculation (Vol. I) will in such cases prevent errors in diagnosis.

**Treatment.** The exclusion of sick animals from work, washing the diseased places with a disinfecting fluid (1:1,000 corrosive sublimate, 2% carbolic acid, creolin, lysol solution, Burow's solution) or inunctions of a 10% naphthalin, naphthol, or salicylic ointment will generally lead to healing in a short time.

Sick animals are to be isolated from healthy ones, and the infected harness and cleaning utensils should not be used on healthy horses without previous thorough disinfection.

**Literature.** Burke, *The Vet.*, 1886, 69.—Dieckerhoff & Grawitz, *V. A.*, 1885, CII, 148.—Friedberger, *W. f. Tk.*, 1880, 413.—Goux, *Rec.*, 1843, 807.—Lührs, *Z. f. Vk.*, 1906, 267.—Schindelka, *Ö. Vj.*, 1883, LX, 61.—Siedamgrotzky, *S. B.*, 1883, 18.—Trasbot, *Bull.*, 1899, 163.

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**Contagious Pustular Dermatitis of Cattle.** Liénaux saw a skin inflammation in a two year old emaciated heifer caused by Preisz's pseudotuberculosis bacillus. It began with the development of nodules, which in time enlarged from pea to hen-egg size and broke open. The loss of substance which occurred in this manner healed here and there, but in other circular places the skin died, and after its sloughing an ulcer with an intensely red base and covered with thick pus resulted; in its neighborhood the skin was raised a certain distance from the underlying tissue. In the surrounding tissue similar nodes or necrotic spots formed,

and the skin became covered with thick crusts of dried pus. Postmortem examination showed no changes in the internal organs. Pustulous inflammation of the skin in cattle can also give rise to inflammation of the lymph vessels; the abscesses thus forming do not, however, break open. The clinical picture then coincides with that of the skinworm (*streptothrix farcinica*) of cattle (Vol. I) (Liénaux, Ann., 1902, 237).

**Other Pustular Inflammations of the Skin.** Kämper and Schumann observed a contagious exanthema in military horses affecting the region of the rectum and perineum and on the under surface of the tail, in mares also on the vulva (Fig. 128). It was not accompanied by itching or by general disturbances of health. Vesicles occurred at these places as large as mustard seeds, peas or



128. Pustular skin exanthema in the rectal and vaginal region of a mare.

one-cent pieces, and after bursting they left unpigmented spots secreting a yellowish fluid. In a very short time these changed to elevations, turned brownish red in color covered with crusts and depressed in the center, which continued to exude a yellowish fluid after the crusts had fallen off. The disease could not be transmitted to healthy horses by inoculation, but Kämper nevertheless was of the opinion that it was transmissible by means of the cleaning utensils. In an enzootic among remounts the disease could be traced with great probability to a cleaning cloth soiled by petroleum or rancid hoof grease. (Kämper, Z. f. Vk., 1903, 440.—Pr. Mil. Vb., 1908.—Schumann, S. B., 1906, 186.)

Scheferling saw an epizootic skin disease, at maneuvers and also in military horses, which could not be transmitted artificially and which was characterized by



vesicles filled with reddish fluid appearing round about the angles of the mouth, and by the loss of hair in the affected places. The formation of vesicles as well as the falling out of hair progressed in the direction of the lymph vessels up to the ear region; aside from intense itching there was acute swelling of the lymph vessels, of the lymph glands and of the parotid region, and disinclination for food. Recovery always occurred in a few weeks.—(Scheferling, Z. f. Vk., 1903, 322.)

In two cases in old Scotch terriers the authors have had the opportunity of observing a peculiar skin disease leading to the formation of multiple abscesses. With the exception of the ends of the extremities the whole of the body was studded with pea to walnut sized hard, painful nodules, which became softer as they gradually increased in size and finally fluctuating. They ruptured early and discharged a thin fluid pus mixed with blood and harboring fine diplococci. The loss of substance healed slowly, and not at all in one case; the open patches gradually increased in size and at their borders the skin was undermined in spots. Owing to the constant new-formation of nodes and abscesses one dog died of exhaustion, the other recovered. Fever was absent in both cases. The disease could not be produced artificially by intravenous and subcutaneous inoculation of the abscess contents.

A similar disease has recently been described by Cuillé (Rev vét., 1905, 750), who proved that the disease was caused by the bacillus necrophorus alone or by this and pyogenic streptococci and staphylococci.

## 16. Hardening of the Skin. Scleroderma.

Gabarret and Lécuyer described a skin disease of swine which occurs especially in old boars and gradually leads to hardening and thickening of the skin. Starting from a spot in the middle line of the back, the process gradually progresses forwards and backwards, as well as on the sides of the body. At first the skin is firm, cool and moist later on, however, it becomes dry, very firm and can no longer be picked up in folds. Together with the gradually increasing thickening of the skin (up to 5 cm. thick) the subcutaneous connective tissue atrophies, the fat layer disappears almost completely, and the shrunken skin is in almost immediate contact with the bones. The animals become greatly emaciated and finally die. The thick skin is extremely hard to cut, the cut surface shows a lardaceous luster and is pure white in color. On cooking it does not become softer, but harder. According to Basset this is a physiological process met with in all boars. (Basset, Bull., 1910, 44.—Lécuyer, J. vét., 1882, 300.)

Pflug saw a similar disease in a calf.

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**Ichthyosis.** (Fish scale disease.) This disease was observed in newborn calves whose skin was covered to a variable extent with horny scales 1 mm. thick, and as a result appeared very rough and stiff. The gray or bluish scales of epidermis formed narrow bands separated from one another by narrow grooves or clefts which were arranged vertically to the axis of the body. The cause of the affection lies in an unusual hyperplasia of the papillae of the skin, which leads to the formation of a very thick and horny epidermis. In the cases which hitherto have been observed the calves lived only 1 to 4 days. In a case noted by Sand the sharp edged scales of the calf injured the mucous membrane of the genital passage during parturition. (Sand, D. Z. f. Tm., 1893, XIX 111.)

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**Acanthosis Nigricans.** (Keratosis nigricans; papillary and pigmentary dystrophia.) This abnormality consists in a symmetrical bilateral hypertrophy of the papillary bodies, increase of pigment in the stratum corneum and germinativum of the epidermis, further, contrary to the like-named disease in man, also in an increase of pigment in the papillary bodies, round about the glands and blood vessels, and finally in a moderate thickening of the horny layer chiefly in the furrows of the skin. This disease was first described in veterinary literature by Schindelka and was recently studied by Halbacher. At the Vienna clinic seven cases occurred in the course of ten years; and the authors have observed three cases. It develops



chiefly in young animals from causes not exactly known. The places of predilection of this skin affection are the axilla, the fold of the stifle, the extensor side of the toes, then the skin of the scrotum, the region around the anus, the under surface of the tail, the lower belly, the under side of the neck, the region of the corners of the mouth, the eyelids and the lips. On these places swelling of the skin and falling out of the hair occurs, the skin being at first softer to the touch than usual, and, in consequence of the more plainly prominent normal skin grooves, it appears like shagreen or seal-skin leather (Fig. 129). With the simultaneous occurrence of a deep



Fig. 129. Acanthosis nigricans on the under surface of the neck of a dog.

grayish blue to black-brown pigmentation isolated papillary or condyloma-like elevations become plainly noticeable, parallel or crosswise ridges form which separate the furrows. In consequence the skin becomes harder and firmer or rough like a grater. Slight exfoliation is noticed only exceptionally, but itching is generally present. At times the process involves the claws or the neighboring mucous membranes, and in such case red, soft, not bleeding, granulations are formed on the mucous membrane.

For the differential diagnosis acariasis must particularly be considered, because in this disease the deposit of pigment in the skin may also occur, but it does not show a symmetrical localization and the abnormal pigment can be removed together with the superficial layer of the epidermis; moreover in acariasis, mites are present in the skin.

The treatment consists in the employment of bran baths with subsequent inunctions of fat, in the internal use of arsenical preparations, as well as in the employment of salicylated alcohol (3%) or of ol. Jecoris Aselli with 2-3% salicylic acid. In the cases of the authors 10% naphthol ointment was used with advantage, while Uebele achieved success with 5% salicylic ointment or with Esterdermasan, provided any granulations that were

present were removed with the scissors. A lasting cure is however not often obtained, relapses being common.—(Habacher, Monh., 1909, XXI, 97 [Lit].)

Pröscholdt found, on the inner surface of the ear of a horse, flat, warty, generally unpigmented, more rarely pigmented papillary acanthomas, which arose from a primary epithelial and secondary connective tissue proliferation. The etiology is unknown.—(Pröscholdt, Papillary Acanthoma, etc., Diss. Bern., 1908.)

## 17. Thickening of Cellular Tissue. Pachydermia.

### (Elephantiasis.)

On the posterior extremities of horses, very rarely on the head, the skin, together with the subcutaneous connective tissue, may thicken considerably as the result of long continued or frequently repeated inflammatory processes (eczema, erysipelatous inflammation, lymphangitis, glanders), or from continuous venous stasis and edema. The affected parts of the body become deformed and the legs resemble the thick cylindrical limbs of an elephant, but if the head is affected it becomes like that of a hippopotamus. On the feet the hypertrophy of the skin and of the subcutaneous connective tissue commences at the coronet

and fetlocks and extends to the legs and even to the thighs. The bending of the joints is more and more limited, and finally becomes impossible; then the horse can only move by flexing the hip joint and dragging its column-like limbs along the ground; if it lies down it is unable to rise without help. In spite of this the animal may be used for work for 1 to 2 years, provided it is not made to go faster than at a walk. The skin is at first rather soft and pits on pressure (Elephantiasis mollis); later on, however, it becomes very firm, almost hard as wood (E. dura). Its surface appears smooth (E. lævis s. glabra) or lumpy (E. papillaris et verrucosa). Kitt observed an actinomycotic elephantiasis of the ear muscles in a pig.

The skin disease itself is incurable and the treatment is limited to keeping clean the thickened parts of the body (washing with Burow's solution or acetate of lead lotion). (Barth, Z. f. Vk., 1908, 60.—Eberhard, B. t. W., 1906, 3.)

### 18. Circinate Ringworm. Herpes Tonsurans.

(*Ringflechte, Kahlmachende Flechte, Borkenflechte, Scherende Flechte, Teigmal, Teigmaul, Maulgrind, Kälber-, Lämmer-, Gaisgrind* [German]; *Dermatomycosis s. Tricophytia tonsurans, Tinea s. Porrigo decalvans; Teigne tondante ou tonsurante, Tricophytie, Microsporose, Teigne de Gruby, Herpès épizootique des poulains* [French].)

Herpes tonsurans is a contagious disease of the skin, which is caused by a thread fungus, the trichophyton tonsurans, and is characterized by the occurrence of more or less roundish, sharply defined spots in the sphere of which the skin is at times covered with vesicles, but generally with scabs or scales, and appears either naked or covered by short hair stumps.

**History.** The trichophyton fungus was discovered in man almost at the same time by Gruby (1843) and by Malmsten (1849) and was named trichophyton tonsurans by the latter author. In a horse the fungus was first seen by Bazin (1853), but the parasitic nature of herpes in the domestic animals was first established in cattle and dogs by the classic investigations conducted by Gerlach (1857, 1859). Hahn (1861) further proved that the ringworm of calves was also of a trichophytic nature; in the cat, Fenger (1861), and in sheep, Perroncito (1872) were the first to find the trichophyton fungus. Further interesting observations and investigations on herpes of domestic animals were conducted by Haubner, Bodin, Bräuer, Siedamgrotzky, Leisering, Zürn, Bodin, Almy & Bodin, Mathis, Pusch, Schindelka, R. Fröhner, Kramareff, Sabouraud, Suis & Suffran, Pécus & Sabouraud, Matruchot & Dasonville, and others.

Shortly after the discovery of the parasitic thread fungus on the skin, some investigators inclined to the view that it was merely a common form of development of the aspergillus glaucus, while Grawitz, who first cultivated the fungus on artificial media, declared the dermatomycetes to be identical with the oidium lactis. Afterwards, however, the



difference between the trichophyton fungus and that of favus was established, and by French authors (Sabouraud, Bodin, Mégnin, Matruchot & Dassonville, Neumann and others), several varieties of trichophyton were distinguished. The theory concerning the multiplicity of trichophytes has been supported by some German authors (Pick, Kaposi, Neisser), but Král, Wälsch, Maiocchi and Marianelli have raised weighty objections which are based on the results of their investigations.

**Occurrence.** Herpes occurs in all domestic animals but most frequently in cattle, next in horses, more rarely in dogs, cats and asses, and least frequently in swine, sheep and fowls; it was seen by Leyendecker in a freshly shot hare. The disease appears to be more or less limited to certain localities, and chiefly attacks young animals. In Germany it is especially frequent in cattle introduced from Oldenburg and Hol-

land, particularly in young stock from Oldenburg (Gerlach, Pusch). In Normandy it is also common. Marshy regions generally appear to be favorable for the occurrence of herpes. According to local conditions the disease is most prominent in stabled animals or in animals out on pasture, yet according to the observations of most authors the complaint is a pasture disease. In this manner the disease, which generally occurs only in isolated cases in cattle and horses, may assume an epizootic extension. Its economic significance is due to the

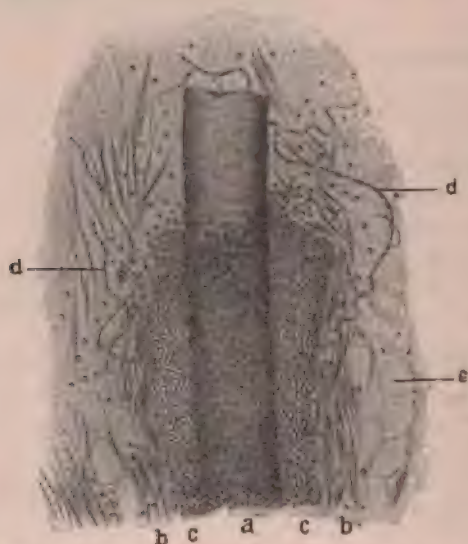


Fig. 130. Trichophyton tonsurans of a calf. *a* hair; *b* remnants of a hair sheath; *c* fungous mantle around the hair; *d* fungous filaments; *e* horned epithelial cells. (Magnif. 200.)

fact that the nutrition of the affected animals is disturbed and that the treatment entails much trouble and expense; moreover, the possibility exists that the disease is communicated to the attendant or to the household of the owner.

**Etiology.** *Trichophyton tonsurans* occurs on the skin of animals in the form of filaments (hyphæ) which are about 4 microns thick, either segmented or undivided; they are fairly uniform and sometimes forked and break up into round or oblong spores (conidia) which strongly refract the light; they may be of yellowish brown tinge and occasionally form chains. The mycelia are found more particularly in the crusts and scabs, the conidia around the hairs (Fig. 130).



**Cultivation.** Cultures of the trichophyton fungus are hard to obtain because it is difficult to isolate it from the germs of other fungi occurring on the skin. For this reason Sabouraud used wort as a medium for the culture of the trichophyton fungus, or a medium composed of four parts of maltose, 2 parts of peptone, 1.5 parts of agar and 100 parts of the distilled water on which only the trichophyton grows luxuriantly, while Král effected the isolation by grinding the hair with pulverized silicic acid and subsequent plate culture. Sabouraud only places the broken up roots of the pulled-out hairs upon the culture medium in order to prevent as much as possible infection with the other microorganisms infesting the hair shafts. Pure cultures may also be obtained by treating the pathological products with potassium hydrate and other chemicals, whereby the trichophyton remains uninjured while other fungi are destroyed (Kitt).

The trichophyton fungus develops on culture media rich in carbohydrates and poor in protein, in the presence of oxygen. For its growth a temperature of 33° C. is most suitable, while above the body temperature the growth is very scanty or ceases altogether; at a temperature of 20 to 24° C. it does fairly well, but in a lower temperature it makes very slow progress (Bodin). Gelatine is liquefied and a substance with ammoniacal smell is formed, and there develops gradually a leatherish, mealy looking, firmly coherent deposit, the under side of which is usually sulphur yellow, while the surface appears white; after long standing yellowish or reddish tints may be seen. The growth of the fungus becomes especially firm and dry on agar (Kitt). On potatoes a wrinkled, fuzzy skin-like covering develops, which is white or yellow, reddish or even brownish in color.

Cultures of the trichophyton fungus like that of the filament fungi of the skin in general are characterized by a rather marked pleomorphism and by great variability, according to their age and origin, as well as according to the composition or age of the culture medium (Král). Considerable differences present themselves not only as regards pigment formation but also the size of the conidia spores. On the surface of the animal body the trichophyton fungus increases exclusively by the formation of so-called mycelium spores, which arise by division of the protoplasm of the hyphæ into four-cornered cells by transverse septa, these cells become rounded off, are detached and after germination again develop into hyphæ. On artificial media spores (so-called chlamydospores) may also be formed by separation of parts of the protoplasm within the hyphæ. But spores also arise by lateral sprouting, and in the filament fungus more or less closely placed pear-shaped inverted projections arise, which soon assume a round form and then are detached (conidia); the conidia-bearing hyphæ appear either in clusters (according to French authors the trichophyton in its limited sense), or they may show a crest-shaped form on account of the closely placed projections (according to French authors the microsporum). Now and then much larger elongated oval, often multicellular spindle spores arise by the development of projections (also called lateral chlamydospores). One also meets with spirally rolled hyphæ in the cultures. The mode of fructification is much influenced by the composition of the culture medium.

On account of cultural peculiarities and clinical differences many authors (Sabouraud, Bodin, Mégnin, Almy & Bodin and others) distinguish several kinds of trichophyton fungi which are said to cause different forms of illness. Thus Sabouraud divided the filament fungi concerned in the production of herpes into two great groups. The



*Tricophyta megalospora* are said to be distinguished in that they form unequal spores, 4 to 6 microns large, arranged in chain-like formation around the hairs, and besides to cause falling out of the hair as a whole, while in cultures the conidia-bearing hyphæ are arranged in clusters. On the other hand the spores of *Tricophyta microspora* (also called simply *microsporum*) do not encircle the hair in chain-like masses, but lie quite irregularly and very close to one another (mosaic-like); besides they are said to be rather small (2 to 3 microns), of equal size, and the mouldy deposit which they form accompanies the hair for some distance outside of its sheath; then the hairs break a few millimeters from the surface of the skin, whereupon the affected parts appear as if clipped short; besides at the points where the hairs break off club-shaped and brush-shaped hyphæ with spores come to view which look like sterigmæ. Finally the cultures are supposed to be characterized by crested conidia-bearing hyphæ.

The *Tricophyta megalospora* were further divided by French authors into several subdivisions to which special forms of disease are said to correspond. Thus *Tricophyton ectothrix* is said to be found exclusively on the surface of the hair roots, *Tr. endothrix* in their interior, while *Tr. endoectothrix* is found in both places. The tricophytes occurring in the domestic animals are classed with the *Tr. ectothrix* or *endoectothrix*, yet Galli-Valerio found a *Tr. endothrix* in a calf, which he identified with *Tr. Sabouraudi*, which is frequently met with in man. The *Tr. mentagrophytes* penetrates the hair bulbs, causes inflammation and suppuration and, in consequence, falling out of the hair. The fungus is said to occur frequently in the domestic animals and the *Tr. epilans*, found by Mégnin in cattle and horses, as well as also the *Tr. caninum* or *Tr. felineum* are probably identical with it. Besides Matruchot & Dassonville distinguish the *Tr. equinum*, Pécus & Sabouraud the *Tr. gypseum granulorum* as special varieties of the *Tr. mentagrophytes* occurring in horses. *Tr. flavum* causes in horses the formation of bald and dry herpes. *Tr. Mégnini* produces herpes of fowls, while *Tr. faviforme* s. *verrucosum* Bodin approaches *favus fungus* culturally, but produces in horses and asses the clinical forms of herpes.

Of the *Microsporum Andouini* which causes in children the *Tricophytia capillitii*, two varieties are differentiated as occurring in animals, namely *Microsporum Andouini* var. *equinum* and the *M. Andouini* var. *caninum*.

Besides these varieties a fungus described by Matruchot & Dassonville is classed with the tricophytes viz. the *Eidamella spinosa* which is characterized chiefly by the formation of perithezia in the cultures and is reported as having produced a herpes-like disease in a dog.

The views concerning the multiplicity of herpes fungi, which are outlined in the preceding paragraphs, were strenuously contested and the investigations of Král, Wälsch, Maiocchi and Marianelli proved that the cultural peculiarities of herpes fungi vary too much, according to their origin and age, as well as the age and condition of the nutritive medium; further that the skin lesions vary too much according to the anatomical structure of the parts of the skin attacked, the variable susceptibility of the animals and the variable degree of virulence of the fungus, therefore the establishment of new species of pathogenic skin fungi is not justified. The differentiation of several species of *tricophyton* has therefore been given up recently and the view is generally being adopted that the *microsporum* and the *tricophyton*, as also their several forms, represent varieties of the same species of fungi which have acquired certain constant and characteristic peculiarities. These characteristics have developed under the influence of the vital conditions which vary greatly, according to the species of affected animal and according to the condition of the skin; they may be transmitted to later generations, and on the other hand the fungi may regain their original form by changes in their conditions of growth.



Nevertheless, especially some French authors maintain the view that the *Tricophyton megalosporum* or the *Tr. microsporum*, as also the diseases caused by them (tricophytia or microsporiasis) differ from each other. But the researches of Král and others are not in accordance with this view, and besides the clinical differences given above are not sufficiently marked to justify, at least for the present, the recognition of two forms of the disease.

**Tenacity.** Fungi that are preserved in the crusts resist desiccation for a long time. Gerlach succeeded, for instance, in producing herpes tonsurans by rubbing crusts into the scarified skin, which had been preserved in paper capsules at room temperature for half a year. Siedamgrotzky and Mègnin could produce the disease with herpes crusts that were 18 months old. Fungous material which was more than two years old, however, remained non-effective (Siedamgrotzky, Duclaux), and in cultures also the fungus had lost its power of germinating after 2 years (Thin). In water the fungous spores can no longer germinate after 8 days, while in olive oil, lard or vaseline they retain their vitality for only two days. They are killed by a 1 per cent solution of acetic acid in an hour, but a one per cent of soda solution or sulphur ointment destroys them only after an exposure of several hours.

**Pathogenicity.** Ground up scabs or hairs containing tricophyton fungi cause the disease after being simply rubbed into the uninjured skin, but the fungi infect much more certainly if the skin is moistened before the application and if the upper layer of epidermis is scraped off or the skin gently scarified. In young animals the first symptoms appear in 8 to 14 days, but if the skin had been moistened or scarified they appear earlier, whereas older cattle are affected somewhat later and are often not infected at all by simple rubbing in of the scab material. If the fungus is placed lineally on the skin, one sees in its wider spread the inclination to rounding off (Gerlach). Artificial transmission is easiest from horses and cattle to cattle, horses and dogs; it is most difficult from sheep and swine to the other species of animals and even from sheep to sheep, and swine to swine. Such transmissions can actually be made in some cases from horse to sheep and swine (Siedamgrotzky, Railliet), from cattle to sheep (Railliet, Schindelka), from goats to cattle (Neumann), from cats to horses and cattle (Williams); also from man to the cat (Fenger) and to other young animals (Horand & Vincens), as also from animals to man (Gerlach, Haubner, Bodin and others). Herpes may also be transmitted from the domestic animals to guinea pigs and rabbits (Bodin, Sabouraud, Friedberger & Fröhner). The presence of pus cocci in the skin checks the growth of the tricophyton fungus (Sabouraud, Marianelli).

**Natural infection** results frequently by immediate contagion, affected animals coming in contact with healthy ones in the stable or still more in the stock yard or in the pasture. Sucklings are often infected while sucking affected mother animals. The transmission may also occur in the covering act; in this manner breeding animals may transmit herpes to entire herds (Pusch). Finally animals may be infected by persons affected with the disease.



Indirect contagion also plays an important part and may be occasioned especially by cleaning utensils, blankets, harness or objects on which the animals rub themselves. Moreover persons may carry the fungus in their clothes or the disease may develop from placing healthy animals in a stable which has previously been occupied by animals that have been suffering from the disease (Schindelka).

Sabouraud and Bodin are inclined to believe that the trichophyton fungus thrives free in Nature on different plants and that animals are liable to contagion on coming in contact with them.

As predisposing causes the following may be noted: housing in warm, damp, dirty stables and particularly basement stables where the animals lie on a thick layer of manure; in such stables the disease frequently remains stationary. Maceration of the skin by getting wet or from frequent washing assists the localization of the fungus, also excoriation of the skin, while exposure to sun and wind, cold stables and living in the open are said to inhibit the growth of the fungus. Consequently herpes spreads much in pasturing districts in fall, in damp summers and especially in winter (Pusch).

The important rôle played by predisposing factors is shown by the fact that one cannot always succeed in transmitting the disease artificially in its progressive form to other animals of the same species, because one is not always in a position to produce the favorable conditions. Thus for instance in one experiment of the authors infection failed to occur in a calf although it was kept for three weeks in constant and immediate contact with a badly affected calf and although macerated scab material had been rubbed on the scarified or shaved skin. On the other hand a sheep suffering from moist eczema on the back and brought in contact with the same subject acquired a general attack of herpes. A dog affected with acariasis was also infected, while another healthy dog escaped.

**Susceptibility.** The cow and the horse are most susceptible to natural infection, then follow the dog and the cat, while the other domestic animals are infected only very exceptionally.

The susceptibility is in so far influenced by the condition of the skin as animals with fine, sensitive hides are easily attacked. According to the investigations of Pusch young animals are for this reason more inclined to the disease than old ones, whose skin is much more resistant, and fine skinned breeds in general are especially susceptible to the disease. The influence of the color of hair on the incidence of the disease may be assumed from the fact that animals with dark hair are more frequently and severely attacked than animals with light colored or white hair.

The affected portions of the skin are not protected after recovery from fresh attacks of the complaint, as the experiments of Gerlach and the clinical observations of R. Fröhner prove (Pusch believes that an immunity after recovery from an attack is not impossible).

There are numerous records of contagion to man through affected animals, especially through cattle, horses and dogs. Principally persons who are occupied with the care, attendance, treatment or milking of sick animals have been infected,



but the infection has also been transmitted occasionally by caressing a dog and by skinning a slaughtered animal which had been affected with herpes. As a result of such infection large outbreaks of the disease have been noticed in man (especially in soldiers). (In the year 1840, an endemic occurred in the Swiss village of Dorkon, whereby in a short time the greater part of the inhabitants were attacked, but Gerlach states that it was due to sarcoptic scabies.) Tricophytia transmitted to man produce an obstinate skin disease causing suppuration of the hair bulbs (Gerlach, Bodin, Pusch, Friedberger and others).

**Pathogenesis.** Herpes fungi infecting the skin grow into the hair follicles and increase between the sheaths of the roots and in the immediate parts; later on they surround the hair roots completely and closely; a delicate plexus of undivided and segmented filaments, and an enormous number of spores being formed, which surround the hair root like a mantel, and often extend for some millimeters above the surface of the skin. Somewhat later the fungi grow through the hair roots upwards and downwards, but the hair bulb remains uninvolved (Wälsch). The fungus also develops in the epidermis just under the horny cell layer.

After the localization of the fungus in the skin, the pathological processes depend upon the virulence of the fungi, the color and anatomical structure of the affected skin, and on the variable susceptibility of the animals. On the sparsely or downy haired parts of the skin, the fungi growing within the epidermis cause a superficial inflammation with scant exudation upon the surface of the skin and proliferative processes in the epithelial layers, and besides an inflammation of the hair follicles and their appendices, through which a nodule or vesicle forms, each corresponding to one hair, while in the region round about the hair the inflammatory process causes reddening, possibly a slight swelling, and always desquamation of the skin (*herpes tonsurans maculosus et vesiculosus*). The irritation of the nerve endings in the skin, which is due to the superficial inflammatory process, always leads to more or less intense pruritus.

On the parts of the skin which are thickly covered with hair the fungi increase in certain forms (generally microsporosis) round about the hair roots, at first in the upper part of the neck of the bulb, and inside the hair root, and also in the epidermis, whereupon the hairs split and become brittle, so that they break off above the opening of the hair bulb (true *herpes tonsurans*); in addition very small vesicles occur but soon disappear, and scaling or desquamation persists (*herp. tons. maculosus*). Itching is absent or only very slight.

In the other and most frequent forms of herpes, on the parts of the skin that are thickly covered with hair (*herpes producing baldness, tricophytia*) which may also occur conjointly with the form heretofore mentioned, the fungi growing in the hair follicles cause folliculitis and perifolliculitis which is not infrequently purulent and varies in intensity; this loosens the connection of the hair root with the hair follicle, and subsequently the hair falls out completely; the hair follicle itself may at times be destroyed and then baldness of the affected part of the skin



by a severe inflammation of the hair follicles and by the exudation of a yellowish, gum-like fluid.

Herpes frequently develops as herpes tonsurans maculosus (true herpes tonsurans) the clinical picture of which was recently described especially by R. Fröhner. Circular spots appear chiefly on the upper parts of the body in which the hair looks as though it were closely cropped. Short hair stumps stand out, surrounded by whitish deposits (fungous mantle) and split at the tips; the surface of the skin in between is covered with scales of variable thickness, which are often glued into crusts (microsporosis of the French authors). The formation of the spots proceeds in such a way that the hair is slightly ruffled up in patches the size of a lentil to a five-cent piece, the skin having a somewhat uneven feeling when the hand strokes it gently, because very small, millet-sized delicate vesicles are present, which, however, dry into thin crusts in a day and which on account of the hair coat remain unnoticed on casual inspection. At the same time there is desquamation of soft, greasy, grayish or yellowish "asbestos like" little scales. In 8 to 14 days, and somewhat earlier in well groomed animals, the hairs break off near the surface of the skin;



Fig. 131. Scab of herpes of the horse.

single hairs, however, fall out whole, and the skin now appears blackish gray for a short time, and somewhat moist, but it is soon covered with thick, profuse scales as described, which unite to form crusts. Meanwhile the spots grow larger at the periphery and become as large as a dollar piece, not infrequently they coalesce with one another, forming irregular surfaces. As soon as the last named size is reached, further spread ceases, scab formation becomes less and gradually new hairs grow which, however, form a contrast to the remaining hair coat by their dark color. Meanwhile, fresh spots always form round about, so that in some rare cases the whole surface of the body may finally be involved. The hairs at the affected spots often fall out, first at the periphery (Neumann) and in consequence ring formed



spots remain for a time, namely until the hairs break off in the center; in very rare cases these ring-shaped spots may be brought about as the result of an early healing of the process in the center (*herpes tonsurans circinatus*). Itching is generally absent or quite insignificant. The disease often occurs in enzootic extension, especially in foals on pasture, very rarely in adult horses (de Doës) (*herpes contagieux des poulains* of French authors).



Fig. 132. Herpes on the head of a horse.

The second form of herpes, which might be designated as *herpes crustaceus* or *trichophytia*, and which is generally caused by the *Trichophyta megalospora*, begins with the occurrence of similar nodular elevations of the skin, with slightly ruffled hair, as in the previous form; in its further course it is distinguished by the gradual formation of irregular roundish or oval, gray or grayish, thick and soft scabs, which may become as large as 10 cm. and adhere rather loosely to the skin; they mat the dry and lusterless hair together, are easily cast off with the matted hair in cleaning and leave completely bald spots behind. The surface of the skin bared in this manner then appears either quite smooth, somewhat moist and still covered by some

hair-root stumps arising from the hair follicles, or a few little vesicles or pustules may be recognized. Not infrequently one observes little depressions with grayish bases on the moderately swollen and reddened skin which have arisen as a result of supuration of the hair follicles (*herpes mentagrophytes*). After the crusts have become detached the slightly moist skin soon dries and is covered for a time with light or slate gray or yellowish scales; through the occurrence of new scabs at the periphery and the resulting falling out of the hair, the ringworm spots gradual-

ly increase for some weeks and then heal up by themselves, while dark hairs often make their appearance. Itching is often present but not of particular severity. As a result of infection in the neighboring parts of the skin the disease extends, so that not infrequently the greater part of the body will be attacked within a few weeks. Evrard, however, saw the formation of nodular herpetic spots over the whole body in one day, while in an enzootic observed by Pécus & Sabouraud the nodules had in the course of two days appeared all over the body in several horses. This occurrence of nodules, which is common in herpes tonsurans of horses, induced the authors to assume the existence of a very special contagious form of herpes (herpes miliaris s. granulosis).

Ringworm develops as *herpes tonsurans vesiculosus* on the sheath and the inner surface of the thighs (Schindelka), in which little vesicles arise in a circular arrangement on the somewhat swollen skin; these, however, soon dry up, the places being then covered by small scales. During the gradual spread of the process in a circular form the individual spots heal up in about 2 to 3 weeks, from the center outwards and the skin easily desquamates.

In asses and mules the trichophytia develops in a similar form as in the horse (Saint-Cyr, Mathis, Neumann, Schindelka) and now and then it is incurable.

#### (b) Herpes Tonsurans in Cattle.

In cattle herpes tonsurans occurs preferably on the head, on the neck and in the region of the anus, less often on the trunk, the croup and on the sides of the chest, while the lower portions of the limbs remain unaffected, even if the disease affects a large part of the body.

The common form of ringworm in cattle is herpes tonsurans crustaceus (scab herpes). It begins with the formation of pea-sized nodules hidden in the hair and covered with little scales, from which flat, raised, sharply circumscribed roundish spots gradually arise, beset with ruffled hair and covered with grayish white and dirty yellowish scales. The spots are at first of lentil size and increase gradually, so that in 10 to 14 days they may be about the size of a one-cent piece and in 6 to 12 weeks they are as large as a tea plate. The skin is then covered by crusts which gradually increase in thickness to the depth of 2 to 7 mm., they are grayish white, resembling asbestos, but appear yellowish on light skin (Plate IV). These crusts attain a considerable thickness (1 cm. and above) especially on dark portions of the skin, while on light pigmented parts of the skin they appear thinner and much more scaly. Together with the formation of scabs the hair becomes lusterless and dry, the dark hairs become somewhat lighter in color, and break off above the scabs,

while white hairs break off very seldom and then only partly, so that the scabs then appear covered with short stumps of hair. The remaining hairs may easily be pulled out on the affected spots.

The scabs at first adhere firmly to the skin, which after their forcible removal, looks as if loosened in texture and bleeds, but later on they are looser, especially in the middle portions, after a purulent fluid has collected beneath them. Besides the purulent fluid, one finds at this stage little pits in the reddened skin, which have persisted, after lifting up of the hair roots and of their thickened sheaths from the suppurating hair follicles (Gerlach).

After 1 to 2 months the scabs generally fall off and leave a bald spot behind, whereupon desquamation continues for a time, and the hairs begin to sprout again. Rarely, and only if the scabs are thin, healing at first occurs in the center, while at the borders the scab is still firmly attached (*herpes circinatus*).

Itching always accompanies the process, especially at the time of development and of healing, still it varies considerably in intensity. Thus Pusch has noted violent itching in Simmenthaler bulls, which disturbed the animals even while feeding, but in grazing animals of the black speckled Holstein breeds itching was almost entirely absent.

Not infrequently the eruption extends, to the parts of the body mentioned above, by the occurrence of fresh spots of herpes in the neighborhood of the old ones (*herpes tonsurans disseminatus*) and a coalescence of the patches is frequently noted where the skin forms folds or where the animal can most conveniently rub itself (head, anal region). Pusch noticed in Simmenthaler bulls an extension of the spots from the tail to the croup and to the flanks, but in this case bald places developed without the occurrence of roundish spots; slight bronchitic symptoms were fairly frequent. By the continual development of new ringworm patches the duration of the disease may be prolonged to six months or a year (Gerlach) and at times it causes great thickening and folding of the skin in Holstein cattle (*elephantiasis*, Köhler, Pusch).

Ringworm sometimes develops in clipped cattle as *herpes tonsurans vesiculosus*, in the form of slightly raised, red, hairless spots which gradually increase in size, a depression forming in the center, and at the same time they become paler and covered with soft lamellous scales, while the margin forms a raised red circle composed of closely ranked nodules and small vesicles. Desiccation of the contents of the vesicles changes these into small gum-like crusts which can easily be removed (Schindelka).

Schleiffer saw a contagious skin affection on the tips of the tail in cattle. It was marked by the occurrence of round spots, redness, vesicle formation and by violent itching; the disease was transmitted to the hands of the attendants, and







was probably trichophytic in origin. Schleiffer, however, claims to have proved that *aspergillus* fungi were the cause of the trouble.

*Ringworm of calves* (Kälbergrind, Teigmaul) is a trichophytic disease of sucking calves in which thick bran-like or dough-like dry scabs develop in the region of the mouth and face, more rarely on the body. The scabs are roundish or oval in form and show hairs with split ends sticking out; they coalesce and finally unite to form an extensive mass of crusts. The diseased spots are sensitive to pressure and violent itching is present, which interferes with the nutrition of the animal.

That ringworm of calves belongs to the ordinary herpes, was proved by Hahn by means of microscopical examinations, and by Schindelka in his animal experiments. (The transmission of the crusts of calf ringworm to cattle and horses caused the usual form of herpes in these animals.)

#### (c) Herpes in the Sheep.

*Herpes tonsurans crustaceus* is rather a rare disease in sheep, which begins with the sudden appearance of circular spots, in size like a one-cent to a twenty-five-cent piece, on the back, chest, shoulders and neck. These spots are recognized by the fact that they mat the wool into tufts and cause it to stand up straight, giving the sheep a tousled appearance. The skin is reddened, often brownish in the center of the spots and covered with grayish white, bran-like rather firmly adherent scales or scabs. The itching is intense, especially at the commencement and causes the animal to scratch and rub continuously, and this leads to a rapid extension of the eruption, with coalescence of the spots and finally extensive baldness (Bräuer, Schindelka).

In lambs herpes manifests itself similarly to the disease in calves, it is called ringworm of lambs (*Lämmergrind*, *Milchgrind*, *milk scab*), but is a rare disease. Thick and dark colored crusts form on the face, especially around the eyes and ears, exceptionally also on the neck. Now and then the disease leads to cachexia and even causes death (Schindelka).

Ringworm of sheep shows a tendency to recurrence (Anacker).

Ringworm has been observed very rarely in goats, in which it occurred as a very mild skin disease.

#### (d) Ringworm in the Pig.

Two cases of the disease were seen by Siedamgrotzky and one by Schindelka. It occurs most frequently on the back and on the outer surfaces of the thighs; also on the croup and the sides of the chest. There are only isolated reddish and roundish spots as large as a twenty-five-cent to a dollar piece, the raised



circular margin of which bears vesicles the size of a pin's head (*herpes tonsurans vesiculosus*); in the center the skin appears normal except for slight scaling. The vesicles soon dry and form brown crusts, while round about fresh vesicles form. In other spots one may see only small, somewhat raised, slightly scaling circles which are reddened at their periphery (*herpes tonsurans maculosus*). The bristles rarely fall out and no large crusts form; itching also is absent. Transmission by mutual contact easily occurs, although artificial transmission to pigs has so far not been successful (Siedamgrotzky, Schindelka).

In this category must probably be classed the skin disease described by Schindelka under the name of *pityriasis rosea*. It affects pigs 5 to 8 weeks old and is accompanied by slight general symptoms (debility, diminished appetite, retarded defecation, moderate tympanites), resulting in recovery in 2 to 3 weeks. On the lower chest and belly and also on the lower surface of the neck, lentil sized, slightly prominent bluish red spots arise which appear to be composed of minute nodules. The spots spread peripherally and become pale in the middle where the skin heals with fine scaling. This otherwise benign affection simulates the like-named skin disease of children (Gibert) which is generally looked upon as of a parasitic nature. Schindelka tried without success to transmit it to pigs and dogs; no fungi could be found in the foci of the lesions.

#### (e) Herpes in the Dog.

The disease generally begins on the head, on the limbs, and on the neck, and not infrequently passes on to the remaining parts of the body, or at times it begins in these parts.



Fig. 133. Ringworm with scab formation in a dog.

The exanthema occurs generally as *herpes tonsurans crustaceus* and is more or less similar to that described in cattle. In the course of a few weeks a sharply circumscribed scaly layer of variable thickness develops, which at first is loose, but afterwards forms a firm scurfy or asbestos-like crust

which cements the hair and gradually grows to a size a little larger than a dollar piece. The hairs become loosened and finally lifted from the hair follicle, remaining fixed in the crusts, and the root ends may be seen on the under surface of the crusts when these are removed. In the further course, according to the species and age of the animals, the skin is either quite bald or studded with many little hair stumps, of copper to brownish red color, dry or moist, thickened or of normal thickness, and bear-

ing few isolated nodules. While crusts form again on some of the bald places, in others only a more or less copious desquamation occurs, hence the process either remains local, or numerous patches of herpes arise, especially when the itching, which always accompanies the process, is intense. If the process extends in this manner, single patches coalesce into irregular surfaces.

A special form of herpes tonsurans crustaceus which hitherto has only been noticed in the dog, manifests itself by the formation of nodular or glandiform semispherical or half-egg shaped elevations on the scalp and especially on the cheeks, 1 to 2 cm. thick, which contrast sharply against the healthy skin (Fig. 133); they are reddish brown in color, rather firm as well as sensitive, while on their surface few hairs or hair stumps stand out. After removing the deposits one sees the widened

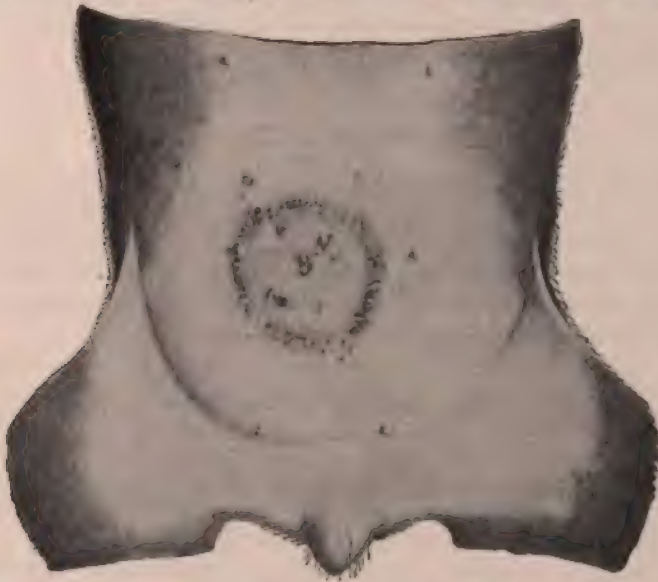


Fig. 134. *Herpes tonsurans maculosus*, on the hairless abdominal surface of a dog.

mouths of the hair follicles, from which pus exudes when the hairs are pulled out or on pressure on the swelling. The complaint is protracted, lasting several months, and after healing the patches remain completely bald (Schindelka).

*Herpes tonsurans maculosus et vesiculosus* not infrequently attacks the parts of the skin which are only sparsely covered with hair and manifests itself by similar symptoms to those described in other species of animals (see Fig. 134). By coalescence of neighboring patches map-like figures may be formed.

Finally a form of the disease described as "*shearing*" herpes (microsporiasis) may be observed very rarely, although according to Sabouraud, Suis & Suffran, it is quite frequent in the surroundings of Toulouse. Bodin & Almy claim that it is



due to a secondary species of microsporum Audouini (*Microsporum caninum*). In this affection fairly numerous disseminated rounded or oval spots are formed, which are sharply circumscribed and are 2 to 5 cm. in diameter. The spots are covered with numerous small, dry, gray scales; the hairs break off in part, about 4 mm. above the surface of the skin, the stumps being split and surrounded with the gray sheaths. Other hairs fall out entirely. Any hairs that may remain in the affected portions may be removed easily and break off close to the surface of the skin. The skin itself under the ringworm spots is either quite normal or it often appears somewhat swollen, painful and on lateral pressure a reddish pus escapes from the mouths of the hair follicles (Sabouraud, Suis & Suffran). Itching is not noticeable. Clinically as well as in respect to its origin, the disease bears a close analogy to the trichophytia of the scalp in children ("tondante rebelle"), but differs from this in that the microsporiasis of domestic animals generally runs a rapid and benign course.

#### (f) Herpes in the Cat.

The cat is infected generally by diseased persons and dogs. The head, the neck and front of the chest form favorite localizations of the disease (Schindelka); the clinical appearances coincide in general with those in the dog, and the disease usually appears under the form of herpes tonsurans crustaceus. Marotel has observed a case of microsporiasis.

#### (g) Herpes in the Fowl.

In birds a few cases of herpes tonsurans have also been seen which, according to Sabouraud, Suis & Suffran really belong to fowl scabies. The affection manifests itself by falling out of the feathers at rounded spots through which the skin is reddened and swollen, especially in the neighborhood of the feathers, the quills of which are occupied by a mantle of wavy fungous hyphæ, which only bear spores here and there.

**Diagnosis.** The recognition of ringworm is generally quite easy. The occurrence of more or less rounded, sharply defined, gradually increasing spots, in the area of which may be seen either short hair stumps and scales surrounded by sheaths or asbestos-like crusts that are often bald or covered sparsely with hair; also the brittleness and looseness of the hairs immediately surrounding the spots, together with the contagious character of the affection, all furnish sufficient points of recognition; moreover, the microscope may be employed for the demonstration of the fungus, although it is not always easy, especially in the cases associated with suppuration or secondary inflammatory changes, as well as in maculous spots.



For the purpose of microscopic examination one scrapes away with a scalpel the epidermis scales or scabs from the most recent lesions, or from the deeper layers of the crusts, and carefully draws out the hair at the margin of the spots, especially hairs surrounded by gray sheaths (fungous mantle). Sometimes the excision of a small piece of skin may become necessary, because it may not be possible to find fungi in the crusts and hairs (Tröster). The material, which may previously be defatted by immersion in absolute alcohol, ether or chloroform, is placed into 1% potassium hydrate solution for a few hours to  $\frac{1}{2}$  to 1 day, according to the thickness of the crusts, and small particles of the scales or crusts and fragments of hairs are examined under 400 to 500 magnification. One may also adopt Sabonraud's procedure of placing only the roots of the hairs on a slide, then adding potassium hydrate and, after putting on a cover glass, warming over a flame until the solution of potash boils, if the hairs are very thick. Besides fungous elements which are arranged in the way already related (see page 900, Fig. 130) one also finds split hairs.

A **macroscopic reaction** of hairs attacked by fungi was announced by Dyce Duckworth and by Behrend, which consists in treating the fungus-containing hairs with chloroform and evaporating, when the hairs appear chalky white, while after moistening with oil their color returns. The reaction is due to the fact that air vesicles collect in the hairs which have become split and teased out by the fungi; after the extraction of the fat the air vesicles reflect the light and in this way cause a whitish color of the hair. By subsequent moistening with oil the original condition is restored.

Favus of sucking animals is sufficiently distinguished from ringworm by the characteristic scutula; the hairs are not split up and brittle in favus, consequently no hair stumps are to be seen on the skin, and after treatment with chloroform no white color appears. In alopecia areata the skin appears healthy, scale formation is absent, and no hair stumps project from the skin. In seborrheic eczema the sharp borders of the spots and the brittleness of the hairs which fall out as a whole are absent. The maculous form of acariasis sometimes causes the whole hair to fall out, but in the fluid contents pressed out from the skin the hair sac mites may be found. Sarcoptic scab is distinguished from ringworm on account of its localization and the appearance of local changes, and besides by the mode of extension and the severe itching.

**Prognosis.** Ringworm of domestic animals is a benign disease in so far as a cure may generally be brought about in 2 to 3 months by appropriate and continued treatment. Spontaneous healing is not uncommon, especially if the eruption is not very extensive. One notices this chiefly in cattle and in adult animals if they are removed from damp stables and are kept in the open in dry, sunny weather. In herds ringworm disappears spontaneously in a short time if the stable is warm and if the herd is small, so that the contagion is rapidly transmitted through the entire stable; otherwise the affection is apt to become stationary. In this connection the character of the disease is not without influence, since in certain years the disease occurs in a particularly malignant form. In young animals (sucking calves and lambs) ringworm is usually very obstinate and causes nutritional disturbances, which may, however, also be noted in older cattle; exceptionally the disease may also cause death. The more extensive the eruption, the more difficult is its cure. In consequence of repeated recurrences and relapses the disease



may be drawn out over long periods, even over one year and longer. The baldness disappears as a rule completely; if it remains it is in ringworm on the face of the dog (Schindelka).

**Treatment.** This is regulated according to the situation and extension of the eruption. As a preliminary treatment, except in the case of short-haired animals and where only quite circumscribed and isolated spots occur, the affected animals should be sheared; but where only a few spots are present, the hair may be pulled out with the forceps or cut away in the affected region and its immediate neighborhood. Hard crusts should be softened with equal parts of soft soap and lard, whether the spots are localized or widespread; in case of an extensive eruption creolin oil (1:20) is most suitable; in this case the fore part of the animal may be anointed with the mixture the first day and the hind part on the following day, and the oil is left on for 2 to 4 days. The softened scabs may then be scraped off or, in extensive cases, removed with fomentations or a bath. Sometimes the inunction must be repeated.

The preparatory treatment is then followed by the administration of antiparasitic remedies which kill the fungi situated on the surface of the skin and immediately underneath, and act thus especially by hindering the further advancement of the process. Anti-parasitics are employed preferably in the form of ointments, for the fungi appear to be peculiarly susceptible to fats and rancid oils (Pusch), and besides the resisting power of the skin against fresh invasions is diminished by fomentations. Mercury preparations, as gray ointment, white and red precipitate ointment (1:10-15), calomel ointment (1:4), corrosive sublimate (1:200 alcohol or sublimate soap) are equally effective, but must not be used on cattle. In cattle and in the other animals, salicylic ointment may be used to advantage, also ointments of tar, naphthol, naphthalin or creosote (1:10) as well as tar or naphthol with soft soap (aa); in dogs Peruvian balsam may be used. In horses R. Fröhner found Baranki's ointment very effective which he had prepared for the first inunction of 1 part of acidum nitricum fumans and 10 parts of adeps lanæ anhydr., and for subsequent applications of 1 part acid nitr. and 20 parts of lanoline. On the other hand Wagner found cresol liniment to give the best results in young cattle (Aqua cresolica, Sapo Kalinus ven., Spir. dil. aa), then creolin soap or creolin ointment. If the eruption is very extensive creolin baths may occasionally be used. In mild cases daily painting with tincture of iodine (1:1-5 alcohol) is frequently successful. All these remedies should be applied not only to the diseased parts of the skin but also to the neighboring parts.

During treatment one should endeavor to prevent the animals from rubbing, consequently exercise in the open, going out to pasture or in the yard is not to be allowed. Finally daily cleaning of the animals should be discontinued. Drying of the

stables by daily sprinkling of gypsum will promote the favorable effect of treatment.

**Prophylaxis.** Even in a slight outbreak of the disease in a large establishment the affected animals should be isolated, and as far as possible placed in a stable by themselves. If this is impossible contact with the other animals should be prevented by leaving an open space between the healthy and the sick animals, by isolating the stalls and by properly tying the animals. Animals suspected of being infected, and especially newly bought animals, should be kept isolated for 8 days; they should not be groomed and should be sheared immediately after stabling. The attendants should be taught the initial symptoms of ringworm with a view to early treatment. Cleaning utensils should be burnt or disinfected, the bedding is to be destroyed or taken away, the stable to be thoroughly disinfected and freely ventilated.

Reuter and Busch advocate compulsory notification of ringworm in cattle, detention of affected animals at the borders, and compulsory treatment, but Fröhner does not consider this to be advisable at present.

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## 19. Honeycomb Ringworm. Favus.

### (a) Favus of Mammalians.

(*Wabengrind* [German]; *Dermatomycosis achorina*, *Tinea favosa*; *Teigne faveuse* [French].)

Favus of mammals consists in the formation of dis-shaped, thick scabs on the skin, depressed in the center, being for that reason saucer or shield shaped. They are sulphur-yellow towards the center and are caused by a filament fungus designated as *Achorion Schönleinii*.

**History.** The favus fungus was discovered in man by Schönlein (1839), and later Remark (1840) gave it the name of *Achorion Schönleinii*, while Gudden (1855) established the fact that the *achorion* fungus was the sole cause of favus. Jacquetant (1847) first mentions favus of the cat without, however, furnishing proof that the disease in question was favus. The occurrence of favus in the cat was first proved with certainty by Draper (1854) and traced to infection from mice



affected with favus; it was again described by Zander (1858). Further reports and interesting experiments have been published by Saint-Cyr (1868), Siedamgrotzky (1872) and Cadiot (1889).

As already related (see page 899) the *Achorion Schönleinii* was first declared to be identical with the *Tricophyton tonsurans*, but later on it was recognized by Grawitz (1886) to be a different variety of fungus, the multiplicity of which was defended in vain by several authors (Quinke, Frank, Unna and others) against the objections raised by Král, Piek, Mibelli, Sabouraud, Tischutkin, and others.

**Occurrence.** Favus is frequent in man, mice and rats; a rather rare disease in the cat, dog and rabbit, while in the cases recorded in horses and cattle (Mégnin, Gigard, Williams) it is not certain that they have not been herpes (Kitt, Neumann). The disease appears as an enzootic, under certain conditions, not only in mice but also in cats (Schindelka).



Fig. 135. *Achorion Schönleinii* from a large crust of a mouse. *a* Mycelium threads and conidia; *b* epidermis cells.

**Etiology.** The *Achorion Schönleinii* is found in the pathological products in the form of glass-like homogenous or granular hyphae, 3 to 5 microns thick, ramifying, tapering at the end; but in places, and especially at the ramifying places, knotty; they appear gnarled and are matted together to form a dense felted layer (mycelium) in the meshes of which the spores, which are usually few, 3 to 6 microns in size, ball, egg or biscuit-shaped, or the conidia of varying size, may be seen (Fig. 135).

**Culture.** The cultural characteristics of the favus fungus have a certain similarity to those of the *Tricophyton tonsurans* (see page 900), yet in this respect there are differences which are not without im-

portance. First of all the favus fungus, contrary to *Tricophyton tonsurans*, needs a large amount of protein for its nourishment while on nutritive media containing only carbohydrates its growth is very scanty. It thrives best at 25° C., although some of its varieties develop at lower temperatures; below 10° C. its growth ceases. The fungus forms, on all culture media, moss-like shoots which start from the periphery of the growth and it inclines almost exclusively to deep growth. On gelatine it forms a deposit which somewhat resembles that of *tricophyton*, but the medium is liquefied only in the course of the second month. On potatoes and beets the growth is perpendicularly raised from the culture. It appears bunched, grayish white and finally grayish yellow. The cultures grown on most solid culture media yield, after short drying (3 or 4 days in the exsiccator), a mortar-like, brittle, yellowish mass. The fungus develops best in 2% meat peptone agar at body temperature (Král). According to their age and origin as well as also according to the nature of the culture medium, the cultures of the favus fungus exhibit a similar pleomorphism and the same variability as was discussed in connection with *Tricophyton tonsurans* (Král, Plaut, Wälsch, Tischutkin). Consequently it is possible for the favus fungus under the influence of different conditions of life, to form varieties which have certain characteristic peculiarities and which are transmitted by cultivation, but which may be changed to their original form by passage through other kinds of skin.

On the ground of cultured peculiarities several kinds of fungus have been distinguished which are said to share in the production of favus. Thus Quinke found three different fungi, described by him as  $\alpha$ ,  $\beta$  and  $\gamma$ . The first and third were transmissible to man and animals (the second was renounced later on by Quinke himself). Also Frank as well as Unna have demonstrated three different kinds of fungi and later on nine kinds were described by the latter author. Sabrazès further maintained that the favus disease of man is caused by the *Achorion Schönleini* and that of dogs by a different fungus, the *Oospora canina*. R. Blanchard also considers man, dog and mouse favus as three different diseases.

As the investigations of Král, Plaut, Mibelli, Pick, Wälsch, Tischutkin and others have proved, the alleged multiplicity of the favus fungus can readily be explained by its pleomorphism. Favus disease consequently must be considered as a uniform disease, which conclusion is supported by the possibility of a mutual infection.

Plaut distinguishes two principal types of the pure culture:

1. The wax type, characterized by yellowish growths of wax-like nature, with radiating folds and central elevations. To this type belong the  $\beta$  and  $\gamma$  fungus of Quinke, *Achorion dikroon* of Unna, Král's fungus, the fungus of human favus Sabrazès, etc.

2. The downy type, characterized by white discs covered with high down, with central irregular elevations; the color is variously snow white, yellow or red. This forms a connecting link between the favus and *tricophyton* fungi. To this type belong the  $\alpha$  fungus of Quinke, the *Achorion cutythrux* of Unna and the organism of mouse favus.

**Pathogenicity.** After artificial transmission, the favus fungus localizes much more readily in young animals than in older ones. Dog favus was transmitted by Sabrazès to man, mice and rabbits; cat favus by Saint-Cyr to cats, mouse favus by Schindelka to dogs, cats and chickens, favus of man by the same author to dogs, cats, cattle, horses and asses.

While the transmission of mouse favus led to disease which was almost like the natural affection in the dog the inoculation of the human favus led in only one case to onychomycosis (the surface of the skin remained healthy), while in cats characteristic scutula developed and on the comb of fowls roundish scaling discs



appeared, in size like a one-cent piece, on the periphery of which small quickly drying vesicles could be seen. In 3 to 4 weeks little sulphur-yellow or whitish yellow nodules developed in place of the discs, and from these elevated hempseed-sized, rough deposits of scales which on falling off left shallow depressions. In cattle, horses and asses, spots were formed like those of herpes tonsurans, and after scraping away of the grayish scales which generally formed, the development of the little favus shields could be observed.

**Natural infection** probably occurs mostly by immediate contact, more rarely indirectly. Obviously cats are chiefly infected when catching diseased mice or rats, which rather frequently suffer from favus. More rarely the disease of cats is contracted from persons affected with favus. Dogs infect themselves in the same way as cats, but besides may acquire the disease from coming in contact with affected cats.

**Susceptibility.** Youth is seemingly an important predisposing factor, as is shown by the transmission experiments of Saint-Cyr and Schindelka. Of equal importance in this respect is the species of the animal, for among domestic mammalians the disease has been definitely demonstrated only in cats, dogs and rabbits. Predisposing causes are probably also found in maceration of the skin and in bad nutrition of the animals.

Transmission of favus from animals to persons has been noted many times (Saint-Cyr, Sherwell, Draper, Horand; Anderson, Smith) and chiefly resulted from fondling, handling or treating sick animals.

**Pathogenesis.** The achorion as a rule localizes in mammals in the orifice of the hair follicle, whence it develops further in the interior of the hair follicle as well as over a large part of the extrafollicular portion of the hair (Unna, Mibelli, Wälsch) and penetrates also between the tissues of the epidermis. On the other hand it seems questionable whether it can pass to the living Malpighian layer, and, still more, to the cutis vera. In the hairs the fungus is found chiefly between the hair cortex and the hair cuticle; it penetrates, however, frequently into the hair cortex without causing splitting up of the hairs. The hair bulb is never attacked or only exceptionally (Unna, Mibelli, Wälsch, Jarisch). The fungous growth gradually leads to the formation of a small sulphur-yellow point covered with epidermis cells which gradually changes to a lentil-sized, elevated, disc-like crust, which is depressed in the center (scutulum—small disc, dish-shaped scab; godet [French]), probably because the development of the fungous colony soon becomes less in the middle of the scab, the conditions of growth being unfavorable there, active growth going on at the periphery (Unna). Except for a few epidermis cells, the crust consists almost exclusively of fungous elements over which the epidermis covering is broken on further growth, and the crusts which develop close to one another become larger by coalescence and enclosing, according to their size, several lusterless, dusty looking hairs. In the immediate neighborhood of the scutulum a wall arises consisting of leucocytes (Wälsch) while the atrophy of the skin tissue



lying beneath the little shield causes a trough-shaped depression to be formed.

On parts of the skin in man which are not covered with hair the development of favus shields is preceded by a process similar to that of herpes tonsurans vesiculosus (favus herpeticus, early herpetic stage of favus). Formerly a similar form of favus, arising from a natural infection, was attributed by Quinke to a special favus fungus (frequently called achorion Quinkeanum) which he claimed to produce honeycomb ringworm in cats and mice.

**Symptoms.** In cats and dogs the paws, especially around the claws, the scalp, and in cats especially the external ear are the favorite localizations of the disease, that is, those parts of the body which are most exposed to infection while the cats play with the mice they have caught; but favus may occur on other

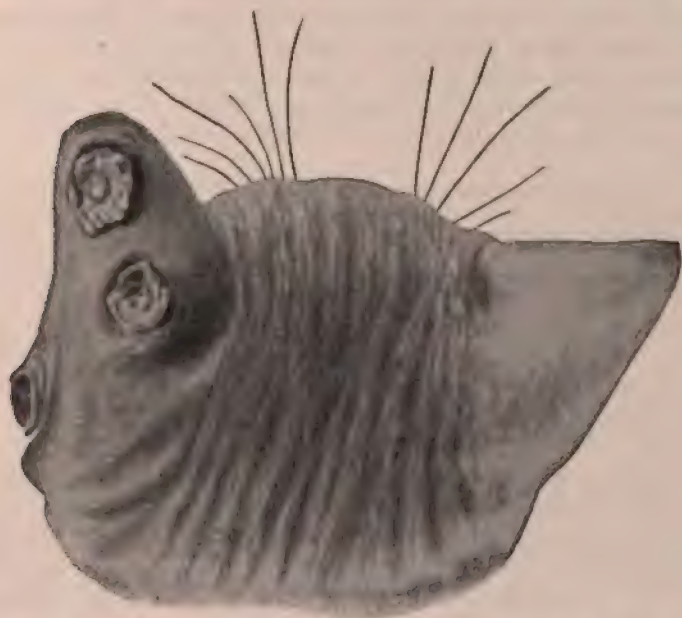


Fig. 136. *Favus scutellum* on the outer surface of the external ear of a cat (after Schindelka).

parts of the body, not rarely in the region of the umbilicus, at the lower chest and on the inner surface of the legs.

Accompanied by a slight or without any itching, small round and yellow or grayish white spots develop here and there over the skin above which they slightly protrude. Gradually increasing in size to that of a one-cent piece, they become rough and concentrically cracked on the surface; the margins are raised, giving the lesions their characteristic saucer or cup-shaped depression. The fungous shields or favus cups (Fig. 136), which are formed in this manner, are yellowish or silver gray on the outside, but always sulphur-yellow in their interior (especially after moistening with alcohol); they may be-

come as much as  $\frac{1}{2}$  cm. thick and from their midst one or several dusty looking hairs arise, which are rather loose and soon fall out. The crusts are removed from the skin with difficulty, and then leave behind a reddened, moist, hairless, trough-shaped depression which, however, is soon covered with a thin layer of epidermis and then has the appearance of a fine glistening cicatrix, the skin appearing atrophic. By coalescence of neighboring scutella, thick deposits of crusts may form which gradually exfoliate in places. At the base of the claws, and still more on the strongly haired parts of the body, crusts do not show their characteristic shape but form only massive yellowish brown or whitish yellow, cracked encrustations (Demons, Neumann). The deposits may easily be crumbled up between the fingers, whereupon a peculiar smell like mouse urine becomes noticeable. The animals also give off a smell, reminding one of cow cheese, if the disease is considerably extended.

If the favus is localized in the neighborhood of the claws these become thickened, loosened and brittle. The disease may also be limited exclusively to the claws (Schindelka).

The microscopic examination may be undertaken in the same manner as in herpes (see page 915). Together with epidermis cells, fat droplets and granular detritus, numerous filaments and spores of the favus fungus are formed in the crusts (Fig. 135) and these are also to be seen in the hairs enclosed in them; yet the hairs are not split and do not become white after treatment with chloroform (compare page 915). In general the fungous hyphae predominate over the conidia in the morbid products, but exceptions may be noticed in this respect.

The alleged **favus of the horse** may occur on the head, chiefly at the forelock and around the eyes, where the crusts unite to form stripes as broad as a finger (Schindelka).

A contagious skin affection was observed by Wilbert in several military horses, which was caused by the *Oospora rubra*, a fungus belonging to the Mucarinæ. At first irregular, roundish, red patches appeared as large as a twenty-five to a fifty-cent piece. The periphery was wavy and covered with a mealy deposit; in places soft scales could be seen. Healing occurred in 3 to 4 weeks after washing with soap and antiparasitic remedies, and after the internal administration of arsenic.

In **sheep** Kowalewsky claims to have observed favus in enzootic extension. In a flock numbering 600 head, 300 animals became affected with an exanthema characterized by dry, grayish white or grayish yellow crusts, which developed on the head, especially in the neighborhood of the lips and nose, and on the inner surface of the external ear. The crusts had a depressed surface and a corresponding bulging underneath and were perforated each by 2 to 12 hairs. The parts of the skin covered by the crusts were moist and reddened. On microscopic examination the presence of hyphae and spores was demonstrated. The general health of the animals remained undisturbed and treatment with carbolic acid cured them in a month. The affection, which, according to Neumann, very much resembles sarcoptic scab, was not transmissible to dogs.

In **rabbits** the disease has been observed repeatedly (Mourraud, Recordon, Méglin, Saint-Cyr). It chiefly affects the head and claws, but any part of the body may be attacked. The process is similar, in

general, to the favus of cats, but may lead to the formation of brittle scabs about 1 cm. broad, of a flattened spherical shape, which on breaking up show a mealy contents exclusively formed by the spores of the achorion fungus. (Mégnin differentiated this disease as favus lycoperdoides.)

Favus of the rabbit often heals spontaneously. Only Mégnin noted one case of death after the anus had been obstructed by a crust that had formed in that region.

**Diagnosis.** In the presence of the characteristic favus shields, honeycomb ringworm may readily be recognized. It might be mistaken for herpes tonsurans, eczema seborrheicum or other skin diseases only if there is no characteristic eruption, as is sometimes the case in the initial stage of favus. In such cases sufficient points to establish a correct diagnosis will be furnished by microscopic examination (scabs almost exclusively consisting of fungous elements, often also the prevalence of hyphæ) and possibly by the yellow color of the scabs after treatment with alcohol.

**Prognosis.** The disease often heals spontaneously and with suitable treatment more promptly; the prognosis is therefore rather favorable.

**Treatment.** After removal of the scabs, if necessary with softening applications (see page 916), the diseased spots may be treated with salicylic ointment (1:10), tincture of iodine (diluted with 1 to 5 parts of alcohol), Helmerich's ointment, corrosive sublimate spirit (0.5%), pyrogallie acid (10 parts to 5 parts of citric acid and 90 parts of fat). The quickest result was obtained by Schindelka by the application of 2 to 5% formalin paste, but considerable pain was caused by this procedure. The remedies are applied repeatedly, the skin having previously been washed; in cats the affected parts of the skin must be cleaned by rubbing with bran instead of being washed.

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**Onychomycosis.** Under this name Ercolani describes a disease of the horn of the hoof that frequently occurs in asses and mules, but more rarely in horses, through which a disturbance of the union between the horny and sensitive lamellæ is caused, leading to the formation of hollow places (hollow wall). In the powder-like substance of the hollow places Ercolani always found a fungus which is called Achorion keratophagus. Baldoni has, however, not succeeded in causing the disease by the artificial transmission of the fungus.



## (b) Favus of Fowls.

*White-comb; Weisser Kamm, Hahnenkamm-Grind* [German];  
*Tinea s. Favus Galli, Tinea cristæ Galli; Favus de*  
*la poule, Crête blanche, Maladie de la*  
*crête, Lophophytie* [French].)

Favus of fowls is a contagious skin disease of chickens which chiefly affects the unfeathered portion of the integument of the head, producing a mouldy deposit, and which is caused by a variety of the favus fungus called the *Lophophyton* (*dermatomyces*) *gallinarum*.

**History.** Fowl favus, which appears first to have become known after the introduction into Europe of foreign breeds of chickens, especially Cochin China and Brahma hens, was shown to be a mycotic disease nearly simultaneously by Fr. Müller (1858), Leisering (1858), and Gerlach (1859), and was later described in detail by Rivolta (1873) and by Mégnin (1881). A thorough etiological investigation of the disease with transmission experiments was made by Schütz (1884). More recently Duclaux (1890), Constantin & Sabrazès (1893), Tischutkin (1894), as well as Matruchot & Dassonville (1899) have studied the disease.

**Occurrence.** Principally fowls belonging to the larger Asiatic breeds are attacked by the disease, especially in zoological gardens, somewhat less frequently indigenous breeds of fowls. According to Sabouraud, Suis & Suffran, cocks are more frequently affected than hens, and fowls with short beaks more than others. The birds are said to fall ill at the earliest in the 6th or 7th months of life after the comb and the wattles have obtained a certain degree of development. Now and then turkeys may be affected (Theobald).

**Etiology.** The *Lophophyton gallinarum* Matruchot & Dassonville (*Dermatomyces s. epidermophyton s. achoxion gallinarum*) is a variety of the favus fungus closely related to the *trichophyton* fungus; it forms two kinds of hyphæ on the skin, the one are long, undulating filaments which are 2 to 5 microns thick, divided into unequally long segments, free from protoplasm, and have delicate walls and often gnarled side shoots; the other are short, at times have forked branches and consist of 3 or 4 thick-walled cells that contain a strongly refractile protoplasm, about 4 to 6 microns thick (by Zürn and others considered to be chains of spores) and later on divide into single cells. The disease is spread exclusively by the last named so-called permanent mycelia, while actual spores are said to be wanting (Matruchot & Dassonville).

**Pure cultures** of the fungus are obtained after isolation (see page 901) at room temperature, but more easily at a temperature of 30° C.

The cultures are distinguished by their slow growth and by the formation of bright red coloring material. In bouillon-peptone gelatine a white mycelium develops with gradual liquefaction of the medium; on a decoction of bread, there forms a faintly glistening white mold.

Constantin & Sabrazès consider fowl favus to be a special variety of favus, while according to Matruchot & Dassonville, Sabouraud, Suis & Suffran, it has greater similarity to the trichophyton fungus. Tischutkin however proved that the fungus changes its character according to its conditions of life just as the ring-worm fungus of sucklings does, consequently he does not consider it to be a special variety.

### Pathogenicity.

If the fungus or scales from diseased birds are placed on the combs of healthy fowls, a skin affection is produced which corresponds to the natural disease. The fungus does not affect the skin of horses, dogs, cattle and cats (Fr. Müller, Gerlach, Schütz, Schindeika, Neumann), while in mice a favus-like eruption and in rabbits and guinea pigs a herpes-like rash has been produced (Constantin & Sabrazès, Sabouraud, Suis & Suffran). In man the fungus causes broad, erythematous squamous spots.

**Natural infection** occurs through immediate contact and also indirectly. Whether the transmission of the disease by the air is possible, as was assumed by Fr. Müller, has not yet been proved conclusively. Early age predisposes to the complaint.

**Symptoms.** Fowl favus begins on the comb and on the wattles with small, white spots like mold, which, under the microscope, may be recognized as scutula; they gradually spread and increase in number, and finally coalesce, covering the whole of



Fig. 137. *Fowl favus*. Mold-like deposit on the comb and upper part of the neck; thick brown crusts on the comb.

the comb or wattles with a white deposit. This gradually becomes thicker, and generally in the course of several months changes into a cracked crust completely covering the comb. The crust may be as much as 8 mm. thick and some parts of it may be dark colored (Fig. 137). Not infrequently isolated, mold-like scales form which enlarge peripherally until they reach the edge of the comb or wattle; they are only slightly thickened, and more so at the periphery than in the middle, by the development of scales.

After a duration of several weeks, and sooner in young animals, the eruption involves the feathered skin of the head, and rapidly passes on to the neck, the back, and finally to the skin of the whole body. This requires several months. The skin becomes thickened at small roundish places and covered with thick scaly crusts (Fig. 137) which are especially strong around the quills and pass down into the feather follicles, whereupon the feathers become loose and fall out. Later on the skin heals in the center and ring-shaped spots develop as in herpes. The roots of the feathers and especially the fine down are surrounded by a white fungous mantle.

Matruchot & Dasseville assert that coincidently with the affection of the comb favus spots appear on the feathered parts of the body, especially on the sides and about the opening of the cloaca.

When the process has involved the feathered parts of the skin, the emaciation of the animal becomes more and more striking. If the eruption attains a considerable extent the animals disseminate a mouldy odor.

**Course.** Favus of fowl often remains limited to the comb and then heals spontaneously. Heim saw a severe case in which the disease was extensive and the feathers had fallen out, and which healed spontaneously after one month. But if the disease once passes on to the feathered portion of the skin it is generally fatal, death being preceded by cachexia and diarrhea. In young fowls the prognosis is always more unfavorable than in full grown ones.

Besides the external characteristic form of the eruption, Schlegel very often found hoarlike and even yellow cheesy deposits, nodules and ulcers in the upper air passages and in the digestive organs, especially in the crop and small intestine, and further isolated foci of necrotic inflammation in the bronchi and lungs. In all these lesions the favus fungus could be demonstrated.

**Diagnosis.** The occurrence of a mold-like layer on the comb and wattles, the gradual spread of the attack, involving the feathered skin, and the contagious character of the disease sufficiently indicate fowl favus.

**Treatment.** Besides the remedies recommended in favus of sucking animals (see page 923), painting with a 2 to 5%



formalin solution may be undertaken and will produce quick results (Schindelka). Painting with benzine, also carbolic acid ointment (1:20), calomel ointment (1:8) and creolin ointment may be employed successfully. If the body is attacked it appears best, however, to kill the bird.

As prophylactic measures sick birds should be isolated, the fowl houses and runs divided, the infected abodes being purified and disinfected.

**Literature.** Galli-Valerio, Schw. A., 1899, XLI, 107.—Gerlach, Mag., 1859, 236.—Leisering, S. B., 1857-58, 32; 1864, 46.—Matrnehot & Dassonville, Bull. de la Soc. mycologique de France, 1899; Bull., 1901, 349.—Mégnin, C. R., 1890, 151.—Müller, O. Vj., 1858, XI, 37.—Neumann, Rev. vét., 1905, 160, 255 (Lit.).—Sabouraud, Suis & Suffran, Rev. vét., 1909, 671.—Sabrazès, Cbl. f. Bakt., 1893, XIV, 152.—Schlegel, B. t. W., 1909, 689.—Schütz, Mitt. d. G. A., 1884, II, 208 (Lit.).—Tschutkin, Die Pilze d. Gattung Achorion. Inaug. Diss., 1894.

**Other Mycotic Skin Diseases in Birds.** Leisering observed an exanthema in a cock, which chiefly affected the region of the cloacal opening; it was conveyed to several hens by the act of coition and caused falling out of the feathers. The points of the feather quills contained a dark colored mass which consisted of numerous dark green or yellow spores.

Rivolta & Delprato noted a skin mycosis in pigeons caused by the *aspergillus glaucus* (*Dermatomyces aspergillina glauca*) in the course of which thin yellow crusts developed all over the body. The crusts were rather broad under the wings, on the lower parts of the body they were thicker, grayish blue in color and smelled badly; on the other parts of the body they were rather small. The birds became much exhausted through the disease and finally died. (Neumann, Rev. vét., 1905, 225.)

## 20. Granular Eruptions of Hogs.

(*Spiradenitis coccidiosa* [Olt], *Hypotrichosis localis cystica* [LUNGERSHAUSEN].)

In granular eruptions small vesicles thickly clustered appear on the skin which, when fully developed, are bluish red or black in color, and therefore give to the skin an appearance as if small shot were imbedded in its tissue.

**Etiology.** The cause of the disease is not yet known definitely. Zschokke, who was the first to describe it (1888) considers that Grampositive cocci, 0.001 mm. in size, enter between the cells of the epidermis and cause proliferation, the formation of epidermis plugs growing inwards, which then coalesce into small vesicles. On the other hand, Olt and later on Voirin came to the conclusion that coccidia penetrated into the efferent ducts of the sweat glands, which become diseased, and that the disease of the hair follicles develops later. The protozoa, which may be seen in the vesicles and to which Olt gave the name of *Coccidium fuscum* on account of their brown color, show ameboid



movement while young. When fully developed they become oval, have a smooth capsule, inside of which a micropyle may sometimes be seen; they are somewhat larger than the *Coccidium oviforme*. Nevertheless Lühe denies that the virus of the disease belongs to the coccidia, although he considers it as probably parasitic in nature.

The mode of infection and the occurrence of conditions influencing the disease are at present not fully known.

**Pathogenesis.** According to Olt the disease process begins in the cells of the sweat glands in which the coccidia appear as brown granules, causing swelling of the glandular cells and rapid proliferation of the contiguous cells. After the cells become detached, they unite with colloid material which develops through the action of the parasites, and with the secretion of the sweat gland, forming a thickish mass. This mass stops up the duct of the gland, which is changed into a small vesicle. When the latter has reached a certain size, its attenuated wall bursts and its contents are poured into the follicles of a neighboring bristle and a larger vesicle develops from the original one after coalescence with the considerably distended follicle.

At first the vesicles situated in the cutis contain a serous fluid and a few red and white blood cells; later on they contain a yellowish brown or quite black tallowy mass which may be stratified and in which generally one or several very fine spirally twisted bristles are imbedded which contain no marrow. These are young and insufficiently developed bristles which have been enclosed in the vesicle and are compelled to roll up in a spiral, in the course of their further growth.

Zschokke explains the development of the vesicles in such a manner that the epidermis plugs become elongated toward the cutis and grow into it, forcing aside its cellular tissue and finally becoming cut off from the epidermis layer. Owing to degeneration of the new formed cells, the foci, which are at first firm, are changed into vesicles surrounded by a layer of epidermis.—Lungershausen believes that the disease is due to a primary faulty development of the bristles which cannot break through the epidermis but remain stuck in the hair follicle, whereupon this is changed into a vesicle, after the outer root sheath has become detached from the inner sheath. Siedamgrotzky, Johné and Ostertag consider the vesicles to be small dermoid cysts; Kitt took them to be atheromata.

**Symptoms.** The eruption begins as a rule on the external surface of the ears, on the croup, on the back, and also on the external and internal surfaces of the thighs, vesicles of variable size and exceedingly numerous being visible in the advanced stage of the disease. The very small ones are pale yellow or white and remind one of pearls by their dull luster; the larger vesicles are of lentil to pea size, hard, lead gray, rust colored or yellowish brown; later on they become violet, bluish red or black, and are only slightly elevated above the surface of the skin. In some places lentil-sized flat elevations may be seen, each with a yellow, brown or black and undulating fine canal which contains one or several bristles. Now and then such a

cavity breaks open and then the bristles lie free on the surface of the skin and nestle there in spiral form. Otherwise the skin does not show any pathological changes.

A watery, reddish turbid fluid escapes from the opened vesicles, and at the same time a brownish thick mass which shows a concentric structure like an onion, together with one or several rolled up bristles; under the microscope flattened epidermis cells are seen in the pressed out contents, also dark brown globules and small round cells.

**Course.** The affection always takes a chronic course and a progressive character. Moreover, once formed, the vesicles are permanent.

**Treatment.** Since from the purely local process no injury to the health has so far been noticed, there is scarcely ever any necessity for treatment. Fomentations with soap water or disinfecting fluids may be undertaken and, besides, the splitting and pressing out of isolated vesicles may be of advantage.

**Literature.** Lühe, Cbl. f. Bakt., 1901, XXIX, 693.—Lungershausen, Über Hypertrichosis localis cystica. Inaug. Diss., 1894.—Olt, A. F. Tk., 1896, XXII, 434.—Voirin, Zool. Jahrb., 1900, XIV, 61.—Zschokke, Schw. A., 1890, XXX, 72.

**Sporozoa-Dermatoses in the Dog.** Marcone saw repeatedly sporozoa-dermatoses in dogs, which manifested themselves clinically in two forms. The one form develops always under the clinical picture of acne, bald spots developing at different parts of the body; the skin is swollen in these places, cyanotic and covered with soft, yellowish gray scales. On the edge of these spots small soft swellings arise and on slight pressure a purulent fluid escapes; often, however, pustules are formed which may be as large as a bean. With a gradual increase in size the spots assume a round shape, the openings of the pustules coalesce into short, red, ulcerous grooves, while the skin is thickened. The organism causing the disease is for the present called *Dermosporidium canis*.

In the second form irregular bluish red and knobby surfaces appear, in the area of which the skin is covered with small glistening silvery scales. On pressure the protuberances discharge a fluid containing mucous-like flocculi. Often fistulous openings are formed, while the skin is thickened and wrinkled. Marcone proposes the name of *Coccidium nudum* for the causative agent, because it does not possess a cell membrane. (Marcone, Z. f. Infkr., 1908, IV, 5.)

## 21. Scabies. Scab.

(*Schäbe*, *Krätze* [German]; *Gale* [French]; *Scabbie*, *Rogna*, [Italian].)

As scab one designates a contagious skin disease which is caused by the mange mites and manifests itself by intense itching and by a more or less violent eczematous inflammation of the skin.



**History.** Scab has been known since ancient times in man as well as in animals; Arabian doctors even mention the scab mites. The view, however, that the disease is caused exclusively by such mites was recognized generally only in the past century. In the 16th century Paré already knew that the parasites bore passages in the skin, and Bonomo & Cestoni (1687) designated them as the true originators of scab. Wedel (1672) discovered the parasites in cats, Kersting (1789) in horses, Walz (1809) in sheep, Gohler (1812) in cattle and dog; finally Spinola (1846) in the pig, and as early as 1791 Wichmann succeeded in transmitting scab from horse to man. In the first half of the last century the etiology of the disease was given a sure foundation by exact clinical observations, as well as by the investigation of the biology of the mites. This is evident with respect to scab of man in Hebra's textbook (1844), with respect to that of the domestic animals in those of Gurlt, Hertwig (1844) and Gerlach (1857). More recent researches (Fürstenberg, Mégnin, Railliet, Johnne and others) have ascertained the part played by the parasites in the production of scab in the individual species of animals.

**Occurrence.** Scab is a frequent disease of domestic animals, and occurs sometimes sporadically, at other times as an enzootic, in practically all countries. The greatest extension occurs in animals living in flocks which are insufficiently cared for.

**Scab of the Horse.** In Germany in the years 1892 to 1908 a total of 10,289 horses fell ill; the number affected varied between 425 (1897) and 959 (1908); out of every 10,000 horses 1.2, 1.6, 1.5, 1.5, 1.6 and 2.2 were affected in the years 1903-1908. The greatest spread of scab in recent years occurred especially on the east Russian boundary in the government districts of Gumbinne, Königsberg, Danzig, Marienwerder, Posen, but in the years 1901-1903 also in Bavaria (Upper, Lower Bavaria, Lower Franconia); in the year 1908 the government districts of Marienwerder, Danzig, Königsberg, Gumbinnen, Potsdam, Allenstein and Breslau were particularly infested.

In Austria in the years 1891-1900, 3,438 horses were affected with scab, the number varying between 190 and 617 (1899). The highest number of cases occurred in Galicia, in which province 2,454 cases were reported. Dalmatia follows next.

In Russia between 1895-1896 there were 4,717 horses ill with scabies.

In Sweden in the years 1892, 1894-1895 there were 193 cases.

In Hungary 35,881 horses suffered from scab in the years 1894-1905; the number of cases varied between 949 (1898) and 5,895 (1901). The highest figures of morbidity were observed in the southeastern counties.

**Scab of the Sheep.** In Germany sheep scab used to be very widespread; in the years 1883-1884, about 260,000 sheep were affected, and of these 78,557 were in Alsace-Lorraine (61% of the total). Since energetic measures of eradication were enforced there has been a decided decrease in the number of cases. In the years 1892-1908 a total of 1,448,122 cases of sheep scab was reported, and the frequency varied in individual years between 53,955 (1906) and 143,468 (1901); in the year 1903 out of every 10,000 sheep 72.56 were affected, but in the years 1905-1908 the frequency per 10,000 was 71.32, 68.24, 102.76 and 132.68. The greatest extension of sheep scab occurred in the government districts of Kassel, Hildesheim, Erfurt in the districts of Jagst and of the Danube.

In France sheep scab apparently does not occur frequently; the greatest extension occurs year by year in the northern, northeastern and the western departments.

In Great Britain the disease is noticed annually in all parts of the country, but it occurs most frequently in the Welsh counties. From 2,000 to 4,000 flocks are annually affected.

*Sarcoptes ovis* is rather common. Most cases occur in the south provinces, but it is also endemic in the other parts of the country.

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The parasitic mange mites on the bodies of domestic animals are divided into the three following varieties:

1. *Sarcoptes* (burrowing mite). The anterior portion of the tortoise shaped or roundish body which is barely visible with the naked eye, measuring 0.2 to 0.5 mm., is formed by the head, of blunt conical shape. This is provided with two strong lower jaws and covered from above with a chest-bead shield which gives it more or less a horseshoe form. The feet are short and thick, the posterior ones do not extend over the edge of the abdomen; in the male cup-shaped suckers on unjointed, fairly long pedicles are situated on the first two and the fourth pairs of feet; in the female on the first two pairs; in the male the third, and in the female both hind pairs of feet end in long bristles.

The following three varieties must be considered:

(a) *Sarcoptes scabiei communis* s. *Sarcoptes major*; in the horse, sheep, goat and man; skin transversely grooved, on the back 6 long-drawn glandiform thoracic, and 14 spear shaped dorsal spines arranged in 4 rows, on the posterior edge of the body four strong bristles (Fig. 138).



Fig. 138. *Sarcoptes scabiei* v. *equi*. Above, left, male. Below, female, from back; right, above, female; below, male, from abdominal side. Magnif. 75. (After Mégnin.)

(b) *Sarcoptes squamiferus*; in the dog, sheep, goat and pig; the anatomical arrangement similar to the preceding, consequently Johne, Kitt and others considered the mite as a variety of the *Sarcoptes scabiei communis*.

(c) *Sarcoptes minor* s. *notoëdres*; attaches itself to the skin of cats and rabbits; body



Fig. 139. *Sarcoptes minor*. Above, female; below, male; both from abdominal side; Magnif. 75. (After Mégnin.)

roundish, without thoracic spines, only 12 dorsal spines; back without scales, skin concentrically grooved (Fig. 139).

The sarcoptes mite penetrates under the skin by the aid of its powerful jaws and head, and immediately digs a tortuous passage above the papillæ of the cutis, in which the female always penetrates further to lay her eggs, while the male takes up his abode superficially in the neighborhood of the opening of the burrow or wanders about on the surface of the skin.

2. *Dermatocoptes* (*Psoroptes*, *Dermatodectes*, sucking mites). These have ovoid bodies 0.5 to 0.8 mm. long, much larger than those of the burrowing mite and readily perceptible to the naked eye. The head is longer than broad, pointed, the lower jaw is straight, suitable for puncturing, the back without spines and scales, with two strong shoulder bristles, slightly grooved, the feet long; all four pairs in the male and the 1st, 2d and 4th pairs in the female end in tulip shaped suckers,



which are placed on long pedicles with three joints, while the third pair of feet in the female terminates in two long bristles; in the male on the posterior edge of the body there are two prolongations, and on the abdominal side two tumbler shaped suckers which in copulation take up the two generative appendages of the female (Fig. 140).

To this class belong:

(a) *Dermatocoptes communis*; in the horse, cow, buffalo and sheep.

(b) *Dermatocoptes cuniculi*; parasitic in the external auditory canal of the rabbit.

The dermatocoptes mites bore through the epidermis layer with their jaws and rostrum as far as the cutis, in order to suck up fluid (hence often colored red); they do not penetrate into the epidermis layer but remain on the surface of the skin, between and under the encrustations.

3. *Dermatophagus* (*Chorioptes*, *Symbiotes*, scale-eating mite); is 0.3 to 0.4 mm. long, its size is midway between that of the two previously described mites; the body is elongated, oval, the head blunt, shorter than broad, bluntly conical; the blunt wedge-shaped lower jaws are only suitable for crushing substances which are taken up; the suckers are goblet shaped, on short non-jointed pedicles; in the female they are found on the first two and on the posterior pair of legs, while the

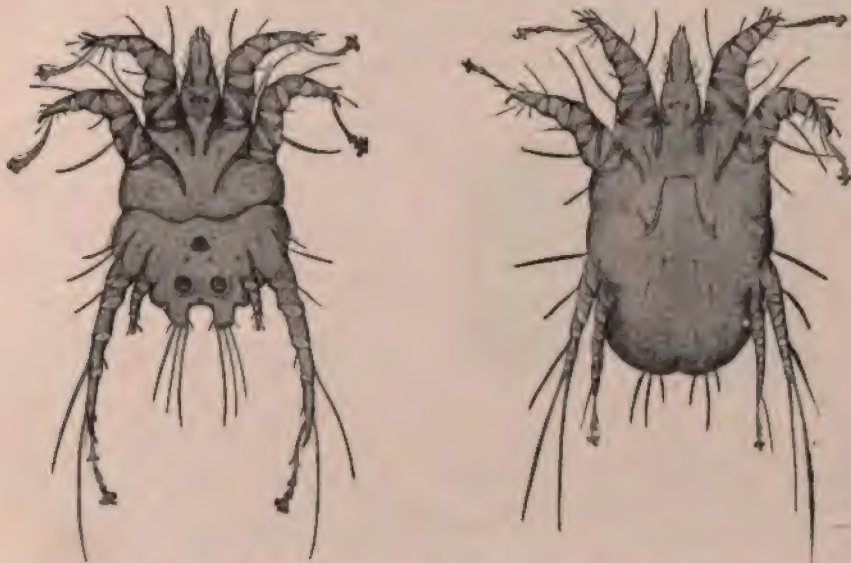


Fig. 140. *Dermatocoptes communis v. equi*. On the right, female, viewed from above. On the left, male, abdominal view. Magnif. 75. (After Ménétriér.)

third pair end in long spines; in the male there are suckers on all four pairs of legs, the last pair is, however, generally rudimentary; the genital plugs or processes are similar to those of dermatocoptes, only with the difference that the plug shaped clasping organ of the male *D. communis* besides the roundish bristles, bears also a rather long bristle, like a blade of grass and slightly twisted.

To this variety belong:

(a) *Dermatophagus communis*; in horses, oxen and sheep.

(b) *Dermatophagus auricularis* (*Symbiotes ecaudatus*); resides in the external auditory meatus of dogs, cats and rabbits (Fig. 141).

The masticatory apparatus of the dermatophagus mite permits them to feed only on the crusts and epidermis scales of the inflamed skin, but not to bore through or dig into the skin.

Besides the enumerated varieties, sub-varieties may occur according to the species of animal on which the mites become parasitic (*Sarcoptes scabiei* var. *equi*, *ovis*, *caprae*, *suus*, etc.); this far-reaching division originated on the one hand through the frequent non-success of transmission, and on the other is founded on differences in size, which, however, probably also depend on the special peculiarities of the body of the host (thickness of skin, hairiness, etc.).

In the individual species of the domestic animals the following varieties of mites occur.

In the horse: *Sarcoptes scabiei*, *Dermatocoptes communis*, *Dermatophagus equi*. The principal form of disease is the sarcoptic scab.

In cattle: *Sarcoptes* (apparently often only after transmission from other animals), *Dermatocoptes communis*, *Dermatophagus communis* (bovis);—principal form, dermatocoptic scab.

In sheep: *Sarcoptes squamiferus*, *Dermatocoptes communis*, *Dermatophagus communis* (ovis);—chief form, dermatocoptic scab.

In the goat: *Sarcoptes squamiferus*, *Dermatocoptes communis*, *Dermatophagus communis* (capræ); chief form, sarcoptic scab.

In the dog: *Sarcoptes squamiferus*, *Dermatophagus auricularis* (canis); chief form, sarcoptic scab.

In the cat: *Sarcoptes minor*, *Dermatophagus auricularis* (felis); chief form, sarcoptic scab.

In the pig: *Sarcoptes squamiferus*.

In the rabbit: *Sarcoptes minor*, *Dermatocoptes* and *Dermatophagus cuniculi*;—chief form, sarcoptic scab.

The **tenacity** of scab mites varies according to the different varieties. According to Gerlach the least resistant are the sarcoptes, which die in dry air in a few days (the sarcoptic mites of the pig after a week [Brandl & Gmeiner]), and in damp media only live a fortnight at the longest. The dermatocoptes mites remain alive outside the animal body 4 to 6 weeks, but longer in damp and dirty stables, and can remain alive even in dry air for 10 to 14 days; Günther succeeded in obtaining a positive result from inoculations of mange scabs kept preserved for 8 weeks at room temperature. The dermatophagus mites remain alive for 10 days, and in damp warm stables for 50 days. From the eggs of mites the larvæ can develop even after several weeks have elapsed.

To the influence of certain chemical substances the varieties of mites show a similar attitude as a whole. They are destroyed immediately by pure or 10% creosote, liquid carbolic acid, wood tar, Peruvian balsam, 30% oil of chloroform, bisulphide of carbon; an almost similar effect is produced after a few (2, 5 or 10) minutes by creolin, liquor cresoli saponatus or aqua cresolica, tincture of iodine, oil of turpentine and petroleum, 3 to 5% watery solution of carbolic acid, concentrated vinegar, ol. animale foetid,

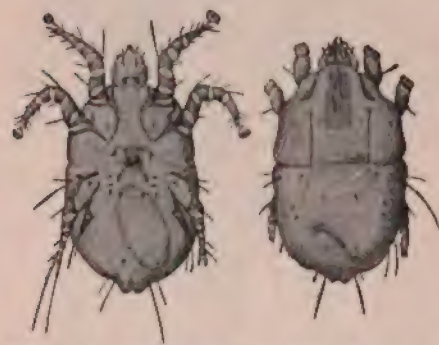


Fig. 141. *Dermatophagus auricularis*. On the left, egg-bearing female from the abdominal side; on the right, the same viewed from above. Magnif. 75. (After Mégnin.)

2 to 3% cargolized oil, 5% salicylic oil and 20% decoction of tobacco, while green soap, spirit of sal ammoniac, gray mercury, 5 to 10% decoction of tobacco, 0.5% corrosive sublimate kill the mite after 1 to 2 or many hours.

With reference to the dermatocoptes mites of sheep scab the investigations of Brandl & Gmeiner show that the mites were killed immediately by chloroform and carbon bisulphide, by 5% carbolic acid in 1 minute, by 2% water solution of liq. cresoli sapon. in 2 minutes, by 2½% creolin water in ½ to 4 minutes, by 1% carbolic water in 4 minutes, by 3% carbolized oil and Peruvian balsam in 8 minutes, by Lugol's solution in 10 minutes, by tincture of iodine in 20 minutes, by petroleum in 22 minutes, by 1% corrosive sublimate only in 50 minutes. In one hour they are not killed by glycerine, rectified spirits, 20% tobacco decoction, 1% watery arsenic solution, gray mercury, 10% corrosive sublimate ointment. Isolated dermatocoptes mites ceased their movements definitely after direct contact with Fröhner's scab dip in 2½ to 4 minutes, with Kaiser's dip in 3 minutes, with Zündel's in 5; but they remained active after an hour in Gerlach's scab dip, in arsenical baths, as well as in Walz's dip.

The **infection** occurs indirectly or by the direct contact of affected animals with healthy ones, whereby either fully developed mites or their larvæ or eggs get on the healthy skin and



here develop protected by the hairy covering or hidden between the folds of hair. The most varied objects, when coming in contact with the affected skin or when touching the encrusted parts, but especially cloths and harness, are frequently the media of infection. Infection by immediate contact happens mostly when many animals live close to or in contact with one another, as for instance sheep, pigs, hounds, further in narrow stables or in horses worked in pairs where the mites can wander from the body of one animal to that of another.

The infection is the easier, the more undisturbed the mites can live on the skin. Among sheep the disease may on this account very easily assume an enzootic character, since the mites can remain undisturbed in the depths of the close fleece. In other animals the development and spread of the skin affection occurs the more quickly the poorer the skin hygiene, and with this the experience coincides, which finds scab the more severe the poorer the owner, while on the bodies of well cared horses it occurs less often, and does not attain such great extension. Here cleaning and curry-combing hinder the settlement of the parasites, and sometimes after a successful infection a portion of the mites are later removed from the skin. Other skin diseases and debilitating influences greatly predispose the body to the complaint.

**Susceptibility.** Scab generally spreads easily and quickly only in animals of the same species, while infection from one species to another occurs much more rarely.

With reference to the direct infection of individual species of animals, and of animals and man, the following was noticed (principally from the statistics of Friedberger & Fröhner):

All varieties of sarcoptes of domestic animals are communicable to man. Sarcoptic scab of man, sheep, dogs, cats, pigs, camels and rabbits is communicable to the horse.

Sarcoptic scab of horses, sheep, goats, cats and pigs (personal observation) is communicable to cattle.

Sheep can be affected with sarcoptic scab by dogs and goats.

Dogs can be affected with the sarcoptic scab of man, pigs, cats, goats, sheep and foxes.

Pigs may be infected by sarcoptic scab of goats.

The sarcoptic scab of man may be transmitted to rabbits.

While therefore the possibility of transmission of sarcopic scab to other species of animals exists, the affection arising thus is sometimes of a mild and transient character, but in other cases severe disease has been noted. It appears that the dermatocoptes and dermatophagus scab cannot be transmitted from one species of animal to another; at most the mites cause transitory itching.

**Pathogenesis.** The sarcoptes mites usually localize on that part of the skin on which they happen to get foothold, while the dermatocoptes mites living on the surface of the skin, and consequently little protected, seek out the more protected parts, or the dermatophages seek out the skin of the extremities or the



anal region. There is probably no doubt that the sarcoptes mites, while boring passages under the epidermis layer, as well as dermatocoptes mites that bite and hold on to the skin, cause an inflammation by their mechanical irritation, which manifests itself by hyperemia of the cutis, exudation, and further sequelæ. Scab mites also produce an especially injurious effect by an acrid juice which they secrete and inoculate into the epidermis and deeper parts, through which sarcoptes and dermatocoptes mites cause an existing inflammation to become more intense. Rubbing in of powdered dead mites as well as extracts obtained from their bodies actually causes itching and soon also the development of papules and vesicles (Bourguignon, Gerlach). Rubbing, scratching and the like caused by the itching visibly increase the aggravation of the inflammatory process.

**Symptoms.** The duration of incubation, the degree and extent of the mange, depend, apart from the variety of mites, very largely on the number of mites present on the skin. Symptoms of disease will be noticed only in the presence of numerous mites; if only isolated mites get on the healthy skin, 4 to 6 weeks may elapse before they have increased sufficiently to cause perceptible changes in the skin, while after an intense infection the first symptoms may become evident after two weeks.

Scab is essentially an eczematous dermatitis. From the bite or puncture of mites transient red spots arise, or at other places nodules, vesicles and pustules and at these points the clinical picture of eczema squamosum develops or more frequently of eczema crustosum with subsequent thickening of the skin. In more severe cases the skin is changed into thick folds, the hair falls out and the surface of the skin is covered with crusts of varying thickness under which the cutis, robbed for the most part of its epidermis, appears bright red, sensitive, moist and bleeding. The pathological changes are the more severe the longer the disease continues; besides the variety of mite has an influence in so far as the sarcoptic mites generally cause a more severe disease of the skin than the dermatocoptes and dermatophagus, which are parasitic only on its surface.

Itching is a constant symptom and it is characteristic to a certain degree that it increases with warmth; in summer it is more intense than in winter; at night or under cover affected animals are more restless than in the daytime and out of doors, and the itching will be more intense when the animals become warmed up by work. This severe itching partly changes the external appearance of the affection, while rubbing, biting and scratching aggravate the inflammatory process, and the continuous unrest leads to loss of condition. Mange declines in cool weather, and in the course of the winter it may appear to be healed, but on the return of warm summer weather it makes its appearance again and assumes larger extension. With

such variations the disease may last for years in horses, dogs and swine.

**Diagnosis.** While scab simulates eczema in regard to the tissue changes which occur, its recognition is possible by its localization on certain parts of the body, by the obstinate itching which increases with warmth, and especially if its occurrence and spread point to an infectious origin. Nevertheless the diagnosis is completely assured only by demonstration of scab mites in the diseased parts of the skin. Examination for mites should not be neglected in doubtful cases, for instance if the complaint is only beginning to develop or has spread over a great portion of the body, so that its starting point can hardly be determined; the exact determination of the variety of mite is generally possible only by this procedure.

The simplest way of **demonstrating the mites** consists in detaching crusts from the diseased skin, especially their deeper parts or still better scrape away the debris of the part of the skin which is covered with crusts and spread it out on a glass plate, watchglass or piece of paper, and afterwards warm it; this may most suitably be done by exposing it to the sun's rays. The warmth causes the mites to move about, and they may be seen on a dark background as small moving points. With the point of a needle they may be put on a glass slide and examined under the microscope. This procedure is suitable for the investigation of dermatocoptes and dermatophagus mites. It is more advantageous to take off the crusts from the skin and put the scrapings in 10% potassium hydrate for 1 to 2 hours, afterwards examining them between glass slide and cover glass, when in the softened and partly dissolved material the uninjured remaining mites or parts of their bodies will be easily recognized under the microscope (characteristics of scab mites see pages 932 to 934). Sarcoptic scab may be diagnosed also by placing the scabs on a man's arm (in 6 to 12 hours itching occurs and pustules form containing minute mites).

**Veterinary Police.** The frequent enzootic occurrence of scab and the considerable losses entailed thereby justify its inclusion in the notifiable diseases. The rules and regulations generally affect solipeds only (horse, ass, mule) and sheep. In solipeds, veterinary police regulations are necessary only with regard to sarcoptic and dermatodectic scab, while they do not appear to be needed against the rather harmless dermatophagic scab. A similar view ought to be taken with regard to dermatocoptic and sarcoptic scab of sheep, although sarcoptic scab is not included under the animal orders of the German Empire. The scab of goats ought also to be included in the veterinary police regulations. On the contrary on account of the very slight transmissibility of sarcoptic scab and the non-transmissibility of other forms of scab to other species of animals the extension of the orders for restriction of trade in regard to other species of animals does not seem warranted. The regulations concern themselves on the one hand with the compulsory veterinary treatment of scabs and on the other with the isolation of affected animals. In the treatment of the horse where the disease is widespread it must not be forgotten that the cost of treatment may exceed the value of the horse, in which case slaughter of the animal will be justified. The skins of

mangy animals should be used in tanneries only after thorough disinfection and complete drying, the wool of mangy sheep should be sold only in enclosed sacks.

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### (a) Scabies of the Horse.

In the great majority of cases scab in the horse is caused by the *Sarcoptes scabiei*, far less often by *Dermatocoptes communis* (var. *equi*). Both forms of scab may spread widely over the body, while *Dermatophagus communis* (var. *equi*) is almost exclusively parasitic on the skin of the extremities.

1. **Sarcoptic Mange.** It begins as a rule on the head, on the sides of the neck or on the shoulders, far less often in the saddle region or on the outer surface of the thighs, but occasionally also on other parts of the body and extends from here over a great part of the surface of the body if the care of the skin is neglected; the extremities, however, are attacked only in very severe cases. As a first symptom intense itching ushers in the course of the disease and causes the animals to gnaw or rub the affected part of the body violently; if one scratches the affected places the animal exhibits signs of pleasure by quivering or turning up the upper lip, by pressing against one and so on.

At first small nodules form on the skin, which may be felt by stroking, and at these places whisps of hair are matted together by loosened epidermis scales, the hair itself being easily pulled out. From the nodules little vesicles develop later on and soon thickish crusts with moist bases. In many cases desquamation alone is noticed for a long time. With the extension of the affection the scales increase, the hair falls out in larger patches (Fig. 142), while in the neighborhood of the irregular bald spots on the skin, which are not sharply circumscribed and are still covered with hair, minute nodules and scales may be found. Later on the skin thickens and thick folds form on the movable parts of the body, on the neck and about the throat; between these the surface appears raw, bleeds easily and exudes a purulent secretion.

In neglected and protracted cases the whole body becomes almost hairless and the diseased skin is covered all over with scabs and crusts; after these are removed a purulent layer be-



comes visible; the thick crusts contain mite burrows. In such cases the condition of the affected animal visibly declines, cachexia may develop and lead to complete exhaustion.

The *sarcoptes* mange extends very rapidly unless it is arrested by painstaking cleanliness and proper treatment, so that the greater part of the body may be attacked in 4 to 6 weeks after the first symptoms make their appearance. Its injurious effect on the whole organism is especially pronounced in old



Fig. 142. *Sarcoptic mange* in the horse.

and poorly fed horses, yet even under favorable conditions it is a serious disease, necessitating energetic and protracted treatment; in severe cases a cure is generally impossible.

Persons, especially the attendants of affected animals, may easily be infected by mangy horses (on the hands and arms or even the body).

2. **Dermatocoptic Scab.** The *dermatocoptes* mite, living on the surface of the skin, prefers the more protected parts of

the body which are covered with long hair, and also the flexor surfaces of the joints. This scab usually begins under the forelock, at the back of the poll, under the mane, at the root of the tail, in the intermaxillary space, in the inguinal region, also on the sheath and udder, exceptionally also in the saddle region or on the croup, and from these places it may spread further on to the body and extremities.

Apart from its varying localization, the symptoms and skin changes correspond with those of sarcoptic scab, and especially in severe cases the two forms of mange are so similar that the differentiation is only possible by a careful examination of the mites. Still the diseased parts of the skin generally appear more sharply circumscribed than in sarcoptic scab (*Schindelia*), and the scabs are generally larger. In this disease also emaciation and cachexia make their appearance.

The development and course of dermatocoptic scab are also similar to those of sarcoptic mange; its prognosis, on the contrary is generally more favorable, for the mites living on the surface of the skin are more easily and certainly destroyed; appropriate treatment leads more promptly to recovery, at least in cases which are not excessively severe.

**3. Dermatophagus Scab.** The disease known as foot scab always begins on the extremities, and only very exceptionally attacks the trunk or the whole surface of the body (Fambach). It mostly attacks the hind feet, less often the fore feet in the fetlock and pastern joints, whence it spreads up to the hock and knee, and sometimes even further, so that finally the thigh and leg, the shoulder and the neck may be attacked. Long fetlock hairs and also neglect of skin hygiene favor the localization of the mites.

Itching is the first symptom also in this disease; the animals stamp their feet, kick, rub the shin bone and fetlock region of one leg with the edge of the hoof of the opposite one and gnaw accessible parts. The itching and the unrest caused by it are noticeable especially at night in a warm stable and are frequently considered as a vicious habit.

The disease of the skin begins with exfoliation and desquamation of the epidermis, which gradually leads to the formation of large crusts and finally, after months, to hypertrophy of cutaneous tissue; large transverse folds form on the pillar-shaped ends of the legs, and in exceptional cases horny and papillous growths may be present.

This form of scab develops very slowly and becomes—in contrast to other forms of mange—more intense in winter, while in summer time it generally improves, of its own accord, to a remarkable degree; with the onset of winter, however, it again assumes a severe form. This is possibly due to the fact that during the cold and damp time of the year a large amount of damp dirt collects on the skin above the hoofs which of itself

maintains an inflammatory process, and in this way indirectly promotes the multiplication of the mites. The disease is comparatively little infectious, being sometimes confined to one leg for a month without passing on to the extremity of the other side. The prognosis is also rather favorable since the disease attacks large surfaces of skin only in very badly neglected animals, and even then it exerts no injurious effect on the general condition of the animal but quickly disappears with suitable treatment.

**Diagnosis.** Although one can recognize mange with absolute certainty only by microscopic examination and this examination seems very necessary in order to distinguish sarcoptic from dermatocoptic scab, yet the clinical signs furnish sufficient points in most cases. Eczematous inflammation together with the characteristic itching always point to scab; severe changes in the skin, and even more a considerable extension of the inflammation which is not sharply circumscribed, as well as marked thickening of the skin will be noticed rather in sarcoptic scab than in that occasioned by the dermatocoptes mites; besides sarcoptic scab is of a very much more obstinate character. In cases that are not neglected, it is generally possible to differentiate the two forms of scab by the localization of the skin changes.

True eczema generally develops at once on a large surface of the skin, the stages of nodules, vesicles and moisture are more pronounced and the itching is only moderate, nor is it increased to any considerable degree by warmth. Pemphigus is also accompanied by extremely severe itching, but the copious serous exudation and the large vesicles are characteristic of this disease. Other animal parasites such as ticks, lice, and exceptionally the *dermanyssus avium* sometimes cause intense itching and later on severe eczema, yet their presence may always be determined by a close inspection of the hair; one should always bear in mind the skin disease caused by the *dermanyssus avium* if coops are found in a horse stable. Dermatophagic scab might be confounded with eczema of the fetlock joint. In this, however, there is no severe itching, and besides the parasites are very easily identified in foot scab.

**Treatment and Prophylaxis.** The treatment differs in part according to the kind of mites present and also according to the extension and severity of the disease. The sarcoptic mites are much harder to destroy than the two other kinds of mites which live superficially on the skin and in and under the encrustations; on this account energetic remedies are generally employed against the sarcoptic mites, and these are applied repeatedly and left for a long time in contact with the skin. On the other hand, the treatment of a very extensive case requires great care and perseverance because the skin changes



are already old, and still more because the animal is generally emaciated and bears the necessary energetic treatment badly.

At the commencement of the skin disease a few applications of gray mercury ointment or of 10% carbolized oil and spirits of tar (āā), with subsequent careful cleaning of the skin, bring about a cure. In cases of longer standing and of a more severe degree of the complaint—the cases occurring in practice are usually of such a kind—the treatment must be much more painstaking.

The preparation consists in thorough cleaning of the skin or in the removal of the crusts and in clipping the hair where possible. The crusts may be softened by smearing soft soap thickly on the skin, perhaps after the addition of 5 to 10% creolin, cresol or carbolic acid; the same effect is produced by smearing the skin with 10% carbolized oil or with 10% creolin, cresol or carbolized glycerine. After 12 to 24 hours the softened and loosened scabs are removed with lukewarm soap water and with the help of brushes, and afterwards the surface of the body is rubbed dry. Next day the actual anti-parasitic remedies are applied either in the form of ointments or of liniments or solutions.

Concerning the means used in treating mange the following remedies deserve to be noticed. Quite fluid medicaments are easier to handle, but they are less energetic than the more concentrated preparations which adhere to the skin for a longer time and at the same time penetrate into the deeper layers of the epidermis, and on this account are more especially to be recommended for the treatment of sarcoptic scab. The selected remedy should always be thoroughly rubbed into the skin with brushes, except on the head, where more cautious rubbing is required in the region of the eyes, mouth and nose. Since most anti-parasitic remedies are more or less poisonous, and the use of a preparation of greater concentration over the whole surface of the skin is not without danger, it appears proper to dress only half of the body at a time, and the other half 1 to 3 days later, after the places from which the medicine may in the meantime have been rubbed off have been dressed repeatedly. An exception may be made in the case of well nourished and strong animals whose whole body may be dressed at one time, provided no creol, cresote, or creolin liniment is applied. The rubbing of the whole body in weak and thin animals should be effected cautiously at three or four different times. On the other hand, washings with alcoholic watery solutions may be applied to the whole skin surface.

Of the numerous anti-mange applications the following are generally used: Vienna tar liniment (pix liquida, flores sulfuris āā 1 part, sapo kalinus ven., alcohol, āā 2 parts; in case the skin is sensitive after the first rubbing 5% of powdered chalk may be added to the mixture). The mixture may usually be rubbed into the whole surface of the body at one time without injury, and may then be left on for six days. After washing and drying the skin the procedure is repeated on 3 or 4 different occasions. Except in very severe cases of sarcoptic scab the treatment always produces satisfactory results. According to Röhl a repeated application is only rarely necessary even in severe cases, but this statement does not agree with general experience. The creosote oil (1 part of creosote in 10 to 30 parts of oil) recommended by Gerlach, or the creosote lini-

ment (1 part of creosote in 20 parts of tar and soft soap  $\bar{a}\bar{a}$ , or 1.5 to 2 parts of creosote, 10 parts of soft soap, and 5 parts of alcohol) may also be employed with very good results; with these remedies half of the body may be rubbed alternately at intervals of six days. With the creolin liniment, according to Fröhner (creolin and soft soap  $\bar{a}\bar{a}$  1 part, alcohol 8 parts), one side of the body may be rubbed on alternate days; after 3 entire rubbings a day of rest is given; repeat 3 to 4 times. The occurrence of somewhat severe irritation of the skin may be avoided by reducing the strength of the applications with alcohol (5 to 7 parts) and any irritation that may already be present is allayed by applying any bland oil. The anointing of the whole surface of the body at one time with creolin liniment may cause the immediate death of the animal (Pr. Vb. 1907). By daily applications with a brush of cresol liniment (aqua cresolica 5 parts, soft soap and alcohol  $\bar{a}\bar{a}$  2.5 parts) and by a thorough cleansing washing on the sixth day, the dermatophagus scab is cured after two of these operations, but the more severe forms of sarcoptic scab necessitate 4 or 5 repetitions (Schlampp). Effective results are also obtained with 5% tobacco decoction, while Schindelka recommends sulphur ointment (flor. sulph. 18 gm., potassium carbon., 10 gm., adeps suill. 120 gm.), 5% creolin (styrax and glycerine  $\bar{a}\bar{a}$ ). The employment of naphthol is not without danger.

The dermatophagus mange is healed in a short time by using 10% carbolic or creolin glycerine, by carbolic soap or tar ointment.

Other parasitocides are: corrosive sublimate (in 2% solution as a wash; only in mild forms and even then not always effective); benzine (5 parts with 10 parts of water), petroleum (with oil  $\bar{a}\bar{a}$ ); both these remedies cause inflammation in the skin, moreover the petroleum has only a weak effect. Styrax as well as balsam of Peru (pure or with 3 to 5 parts alcohol) are only suitable for applications to small skin surfaces on account of their high price. Arsenic (1% solution in vinegar or as Viborg's bath; Acid. arsenicos.). Potass. carbon,  $\bar{a}\bar{a}$  1 part, Aqua, Acetum comm.,  $\bar{a}\bar{a}$  100 parts; of the solution at most 500 gm. at one time), potassium sulphide (with 5 to 10 parts of water), etc. Fürthmaier treated horse mange successfully with a formalin soap solution in the following way: 100 grammes of soft soap are dissolved by warming in 750 gm. of water, after cooling, the fluid is decanted, and after the addition of 100 gm. of formalin (40% formaldehyde) it is filled up to one liter with lukewarm water; applied at first only to the diseased places but afterwards also to the healthy parts of the body; repetition after 5 to 6 days; in obstinate scab 3 to 4 times, but the last two applications are to be made at intervals of 8 to 10 days; after rubbing in there was transient unrest. Recently in Germany a 25% watery solution of therosot which contains mercury has been largely used (apply to a third of the body at a time and afterwards blanket the animal; repeat after weekly pauses). Washing with a 4% bacillol solution has caused transitory symptoms of poisoning and falling out of the hair (Hennig).

During the treatment good and plentiful food must be given to the horses, and they must be kept in dry, clean and moderately warm quarters. Since in spite of careful treatment single mites may remain alive, it is well to watch the animals for some time, and to resume treatment immediately on the occurrence of suspicious symptoms. In this case less heroic remedies may be employed successfully. It is especially necessary to watch

the animals at the commencement of the warm time of the year, since mange may remain latent all winter.

The sick animals must be isolated, and the healthy ones must be cleaned carefully and kept under observation for a long time, for infection cannot always be excluded by the most careful examination. At the same time a thorough cleaning and disinfection of the stable, blankets, cleaning utensils and such like must be carried out.

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### (b) Scab of Cattle.

*Dermatocoptes communis* (var. *bovis*), and still more frequently *Dermatophagus communis* (var. *bovis*) attack the body of cattle, while *sarcoptes* mites have hitherto been found only rarely. In North America dermatocoptic scab seems to be much more frequent (Hickman).

1. **Sarcoptic Scab.** This form of scab seems mostly to arise by transmission from the horse (Froriep), from the goat (Röll), from the dog (Viborg), from the sheep (Pflug) as well as from the pig (Marek). It affects chiefly the skin of the head and neck, and according to Röll quite similar symptoms are exhibited to those of sarcoptic scab of the horse (Fig. 143). In a case observed by Wolffhügel, however, this mange affected the whole surface of the body with the exception of the head and neck, and the wrinkled, bleeding and leathery skin was covered with scabs  $\frac{1}{2}$  cm. thick. In one of the authors' cases the disease occurred on the fetlocks as well as in the fold of the stifle and manifested itself by inflammation, nodules, and scale formation; owing to the severe itching the animal very often licked the affected parts. The hair fell out at the places mentioned. The disease may be of short duration and may heal spontaneously.

2. **Dermatocoptic Scab.** This disease is on the whole not very frequent; after infection through diseased cattle (cattle are not infected with *dermatocoptes* mange by horses and sheep) it begins on the upper edge of the neck, at the foretop, at the base of the horns, at the withers, on the sides of the neck, another time at the root of the tail. It preferably advances in the direction of the long axis of the body, later, however, spreads also along the transverse axis and passes on to the lateral surfaces of the chest and belly, while, even in severe cases, the legs remain unaffected. The intense itching causes the animals to lick their bodies frequently and to rub themselves on fixed objects; little nodules form in the skin and soon afterwards brown crusts, which in time attain a quite considerable thick-



ness. At the same time the skin becomes bald, like parchment or leather, stiff and wrinkled, but its surface is dry even under the crusts, and in the neighborhood it is covered with bran-like epidermal detritus. The severe rubbing may modify the clinical picture to such an extent that the accompanying dermatitis may cause the skin to be raw and even ulcerous. Left to itself, the process spreads gradually and not by leaps and bounds as is the case with sarcoptic scab; meanwhile the animals fall away in condition, they move stiffly, cows fall off in their milk supply, and finally the increasing cachexia sometimes leads to the death of the animal. At times the disease improves spontaneously with the passage of winter, the crusts fall off, the hair grows again partially, but the following winter the affection recurs if the animals are kept in warm, damp barns, and then it assumes a severe form; thereafter alternating improvement and aggravation dependent on the time of the year may repeat themselves several times (C. Müller). The disease also occurs in buffaloes and produces similar symptoms (Röll).

**3. Dermatophagic Scab.** This develops most frequently in the grooves at both sides of the root of the tail where, with symptoms of moderate itching, fine, dry, bran-like scales accumulate. Sometimes the skin looks at first as if dressed with varnish (Kaiser). Mange may also be observed in the fold of the fetlock (foot scab) where it resembles eczematous "greasy heels," but on the whole it is then a benign disease (Rabe). Only in badly neglected cases it spreads from these spots of predilection to the croup, loins and back, on the perineum, the inner surface of the thighs, sometimes also to the scrotum or udder.

Apart from these rare cases in which also the condition of the affected animals suffers, the disease is generally benign and may even remain stationary for a year without injurious results. Its infectiousness is also slight. In the cold season the disease generally gets worse.

**Diagnosis.** All three forms of scab, especially in their incipient stages, may easily be recognized; the demonstration of the mites also presents no difficulties, for they are generally present in great number between the crusts and scales, while the dermatocoptes mites are generally present at the periphery of the diseased surfaces where the disease products are fresh.

The *Trichodectes scalaris* or the *Hæmatopinus eurysternus* also causes itching and superficial eczema, but their presence may be easily recognized with the naked eye (it is to be remembered that these parasites may also occur in mangy animals). Idiopathic itching of the skin is differentiated from mange by the acute onset and by its extension over considerable portions of the body, and also by the negative result of microscopic examination, while for eczema the more acute symptoms of in-

flammation, and especially the moist surfaces, are characteristic, in contrast to the dry crusts and dry skin surfaces in scab. Ringworm is recognized by the round, gray or yellow coherent crusts and by the fact that the hair falls out in round patches.

In view of the diversity of the parts of the body which are first attacked, the differentiation of the three forms of mange from one another can only present difficulties in very old cases, although the dermatophagus scab never attacks the whole body.

**Treatment and Prophylaxis.** After careful cleansing of the skin, and if possible after clipping of the hair, the following



Fig. 143. *Sarcoptic scab* in a calf.

remedies may be employed best in fluid form, and applied at most to one-fourth part of the body: 5% creolin-, lysol-, or lysoform solution; naphthalin ointment (1:10); creosote (1 part to 10 parts of alcohol or 15 parts of water, or 1 part of creosote to 15 parts oil); petroleum (with 3 parts of water or an indifferent oil), 5% tobacco decoction, perhaps also sulphur ointment (1 part to 4-5 parts of fat); all of these applications must be washed off with soap and water after 3 days. In North America the treatment by dipping is frequently employed (Hickman). Mercurial and arsenical preparations are generally to be avoided, as they easily cause poisoning. Finally the dermatophagus scab

may suitably be treated with creolin or tar liniment, in case it is only limited to the neighborhood of the rectum or fetlock.

Disinfection of the stables and fittings is naturally also necessary in order to secure success.

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**Mange in the Camel, Dromedary and Llama.** In Africa sarcoptic scab is frequently observed in camels or dromedaries, and in zoological gardens the disease may likewise be seen in these animals and in the llama. The cause of the disease is the *Sarcoptes scabiei*. The affection begins on the parts of the body which are covered with thin skin (inguinal region, the lower abdomen and the axilla region) and spreads from here to the body and neck, to the extremities and to the tail. Together with intense itching, nodules and later on crusts and thick scabs form. At the same time the hair falls out, while the skin gradually thickens and becomes wrinkled. If timely treatment is not adopted, the skin becomes cracked as a result of continuous rubbing and often ulcerous patches and secondary infectious or phlegmonous inflammation of the subcutis occur. The disease spreads very quickly in a herd, especially in spring (Cadéac), and affects principally the young and the very old animals. The mites are also transmissible to man (Piot).

The treatment is similar to that in scab of cattle; but balsam of Peru is especially recommended (Johne). The animals must previously be clipped (Johne noticed fatal poisoning after rubbing in of liq. cresol. sapon. 1:4 on all four extremities and on the lower abdomen.

### (c) Scab of Sheep.

The most frequent disease of sheep, which is usually called sheep scab, is caused by the *Dermatocoptes communis* (var. *ovis*), while mange caused by the *Sarcoptes scabiei* (var. *ovis*) is much less frequent; dermatophagus mites occur only exceptionally on the bodies of sheep.

1. **Sarcoptic Scab.** It occurs exclusively on the parts of the body which are not covered with wool, beginning generally on the lips, in the neighborhood of the angles of the mouth and on the edges of the nasal openings, from where it extends to the other parts of the face and to the external surfaces of the ears (head scab of sheep); in exceptional cases, especially if they are severe, it may attack the feet at the back of the carpal and tarsal joints and also spread further downwards.

The itching is intense and the animals rub their mouths vigorously on the rack, on the ground and on the fore legs. Little nodules and occasional vesicles form on and around the lips, and in these places crusts are formed, which are at first thin, and later on rather massive, gray, dry and firmly adherent. At a later stage of the disease the whole face may be cov-



ered with a cohesive layer of crusts and at the same time the skin swells as a result of frequent rubbing, becoming cracked and raw and increasing the swelling of the face.

The disease is benign inasmuch as it soon heals under treatment; if, however, it is neglected, the severe swelling of the lips may hinder the taking of food. Now and then the affection may be transmitted to man (Gerlach), and on the other hand intermediate infection may occur from mangy goats (Wallraff and Roloff).

According to Roloff & Neumann, *sarcoptes* mites avoid only the skin that is covered with oily wool, while in sheep with wool that is not fatty (Hungarian, Neapolitan, fat-tailed sheep) they also settle on the trunk and may even spread over the whole body; in this case only a few large, hard and armor-like crusts form on the skin.

**2. Dermatocoptic Scab.** This form of scab is very frequent as a flock disease and occurs exclusively on the parts of the body which are thickly covered with wool, where the mites are well protected and have sufficient warmth so that they are able to multiply rapidly. It begins usually in the inguinal region and on the back, spreading from here along the back to the sides of the body; in rarer cases it establishes itself here first; the skin in front of the chest and on the lower abdomen is never attacked.

At first one perceives unrest caused by itching, then rubbing and gnawing of the posterior half of the body; soon the wool is loosened at isolated places and single locks of wool are raised over the surface of the fleece. When the diseased places are scratched or rubbed with the hand the animals show a feeling of pleasure by stretching the head, moving the lips, nibbling with the teeth, pressing against the scratching hand, movements of the tail and scratching with the hind feet. The itching is most severe in the warm summer time, after exercise, in the barn and at night.

On parting the wool one encounters lentil-sized yellow or reddish nodules on the skin, perhaps also little serous and purulent vesicles may be seen, which dry up together with their contents and change to thin, fatty, yellowish scales. By the occurrence of fresh scales in the immediate neighborhood and the increasing exudation, fatty yellowish crusts and scales gradually form. With the further extension of the process the small spots unite to form larger, irregularly-shaped surfaces, which now become covered with very thick, yellow crusts. These mat the wool and are lifted up by the growing hair. As a result great tufts of wool are thus raised from the fleece, and the surface of the fleece becomes uneven; larger or smaller bunches of wool may fall out, whereupon bald surfaces arise on the body, especially along the line of the back, which are now covered with dry scabs. The skin then appears thickened, stiff and cracked in places. In shorn sheep the crusts are dry and brown in color on account of plentiful evaporation (Fig. 144).

The changes extend quickly under the long fleece, especially in stabled sheep, so that within 6 to 8 weeks the whole of the body may be affected. In infected flocks the development occurs more quickly, because by the near contact of the equally contaminated fellows fresh mites are always transmitted to the affected bodies, and these now attack fresh areas which soon coalesce with those already present.

In consequence of the softness of the skin, and also on account of the thickness of the fleece, young well-bred sheep are generally infected more easily and attacked more severely. If the disease has already attacked the greater part of the body, emaciation sets in, and the increasing anemia and



Fig. 144. *Dermatocoptic scab* in the sheep.

cachexia finally lead to death. Such an unfavorable result is, however, usually observed only in winter when the animals live huddled together in warm stables, while the disease quickly subsides in summer after shearing and when they are in the open.

**Dermatophagic Scab.** This form of mange occurs very rarely and is only slightly contagious. It always begins on the hind fetlocks and their joints (leg scab, fetlock mange) where the skin becomes inflamed, covered with fine little scales, and later by thick crusts, finally becoming thickened and cracked. The animals stamp their feet and gnaw the affected foot. The affection only exceptionally passes over to the forelegs and



may rarely extend upwards to the scrotum or udder; with the onset of spring it subsides spontaneously to a striking degree.

**Diagnosis.** The comparatively large dermatocoptes mites may be easily found, especially on the recently affected places, under the damp crusts; while in older foci where the crusts are hard and the skin under them has lost its elasticity, the mites are only few. Prior treatment naturally makes the demonstration of the mites difficult. For further data on the demonstration of the mites see page 937.

On account of the intense itching the disease might be mistaken for trembles, and the more so as in this disease the skin may become eczematous in consequence of rubbing; yet the other symptoms of the disease (terror, motor disturbances) and the absence of mites on the inflamed skin ought to render the differentiation possible in every case. Mites are also missed in eczema which occurs after getting wet through or when out in the open in the cool autumn time; it is much easier to heal and is not accompanied by such intense itching. In certain cases also the exclusion of seborrhea (so-called false scab, tallow mange) might present difficulties, because in this complaint whitish yellow fatty scales or lamellæ form, and later on the wool may fall out, or at times the animals pull it out. In contrast to scab, however, the skin appears normal in seborrhea, intense itching is absent, and the disease does not spread in the flock, although the simultaneous affection of several animals is quite possible if they are fed unsuitably. Itching also exists in the presence of parasitic insects (*Trichodectes*, *Melophagus*) or ticks, yet one can see these with the naked eye between the hairs of wool, and the skin appears uninjured thereby.

The shepherds often seek to make the scab of sheep unrecognizable to a certain degree by the examining veterinary surgeon. The pleasure exhibited by an affected animal on scratching is sought to be suppressed by pressing the sheep to be examined between the thighs. By some remedies that may have been employed the discovery of the mites is rendered difficult, and perhaps a severe inflammation of the skin may be produced, which on scratching is manifested rather by signs of pain. Such applications (so-called grease spots, or grease plates) should always awaken suspicion of scab and lead to a careful examination.

The different forms of scab may easily be distinguished from one another according to the parts of the body attacked, quite apart from the fact that the dermatocoptic scab is far more frequent than both other forms.

**Treatment and Prophylaxis.** On account of its frequency and economic importance, dermatocoptic scab is most frequently the object of treatment and is in itself cured easily enough, but the carrying out of the treatment in practice is troublesome, since many animals must be treated at once and one must be particular to protect the wool wherever possible. Since it is not always possible in a scabby flock to separate the com-



pletely healthy from the already affected sheep, because single mites under the wool or even little nodules or crusts may be easily overlooked on the most careful examination, the disease may be exterminated only by treating all the animals in an affected flock by dipping or washing, or in mild cases of disease by smearing.

The radical treatment by means of dipping can only be undertaken in the warm time of the year because of the danger that the animals catch cold afterwards, and all the more so as the sheep generally must previously be shorn. In cases where the disease occurs in the cold time of the year, and if the disease is only slight, the visibly diseased animals should receive preliminary treatment in order to prevent or hinder the spread of the complaint, and with the onset of the warm time of the year thorough treatment may be given to the flock.

The treatment by **inunctions**, which is adopted in ordinary sheep scab, is only palliative; it consists in rubbing the diseased parts of the skin at times with anti-parasitic ointments, liniments or oils. For this purpose are suitable applications of 10% carbolated or creosote oil, creolin-, lysol-, cresol- or bacillol liniment (1 part of creolin, lysol, liquor cresoli saponatus or bacillol, 1 part of alcohol, 8 parts of green soap), petroleum, Helmerich's ointment, oil of turpentine, nicotine, and gray mercury ointment (only to be employed on a few animals, when these do not come in contact with lambs). Ostertag succeeded in eradicating scab from flocks by inunctions with 5% of oil of turpentine added to the juice of pressed tobacco, applied to all suspicious places; this treatment was repeated 2 or 3 times at intervals of 7 days. On the other hand the observations of government veterinary surgeons in Germany lead to the conclusion that the use of inunctions exerted an unfavorable influence on the total results in by far the great majority of cases of scab.

The only thorough cure is by **dipping**, and is best carried out in warm weather. This is preceded by shearing of the sheep and by the usual simple water bath, not only in order to utilize the wool, but also in order that the remedies employed may come in intimate contact with the diseased surfaces. In case of necessity long fleeced animals may be treated, yet here the result is uncertain, although Gmeiner, Grün and others consider that dipping the sheep in such cases is advantageous, because the dip being stored up for days in the fleece can exert its effect for a much longer time. After shearing it is advisable in severely affected animals to soften the massive crusts with soft soap, or better, to rub in a parasiticide immediately, such as creolin, bacillol-, cresol- or lysol-liniment, and on the next day to scratch off the crusts with a wooden knife or wash them away with soap and water. (In Prussia this preparatory smearing is especially recommended before dipping in cresol preparations.) The dipping then follows 3 to 4 days later, in the case

of arsenical dips after 8 to 14 days, but the dip must be repeated once or twice after an interval of 8 days, especially if cresol preparations are employed.

**Dips.** While formerly rather complicated mixtures were employed which stained the wool more or less, simpler applications are now used which save the wool as much as possible. The following are used:

*Dips Containing Cresol.* The dipping fluid is a 2 to 2½% watery solution, which is prepared by adding for every 100 sheep 5 to 6.75 liters of purified creolin Pearson (Fröhner), or lysol (Maisel), or liquor cresoli saponatus (Brandl & Gmeiner), or bacillol (Paszotta) to 250 liters of water, which should be poor in lime, and mixed thoroughly. The advantage of cresol baths lies in their simple mode of preparation and in their cheapness (the cost of a bath is about \$2.00), also in the fact that the wool suffers no injury. On this account baths containing cresol, but especially Fröhner's creolin bath, are at present most frequently employed in cases of scab.

The dipping procedure is as follows: one of the mixtures mentioned is heated to 36 to 38° C. and poured into a wide bath tub; near this it is well to place a second shallow bath. The sheep are then bathed singly in such a manner that a man seizes the fore and hind legs and another the head of the sheep, at the same time closing the eyes of the animals with his thumbs, whereupon the sheep is dipped into the fluid with its back downwards and held there for 3 minutes in such a way that only the nasal part of the face is above the level of the dip. One must be careful that none of the fluid is swallowed. Then the sheep is placed on its legs in the shallow rubbing off tub close at hand, and is brushed, kneaded, scratched and worked all over the body by two other men, especial attention being paid to the visibly diseased parts of the skin, and the crusts being removed as far as possible. This being done, the sheep is again dipped in the bath fluid and then set free. In woolly sheep one limits the operations to working on the skin, unravelling the matted wool and running one's hand through the fleece. In ewes the udder should afterwards be washed carefully with water.

The dipping operations should be carried out in a place that is free from draughts, clean and sunny, and the sheep should be protected from cold and wet until they are completely dry. If some sheep show symptoms of scab after the second dipping 8 days following the first, the whole flock will have to be dipped a third time.

The Prussian regulations of the year 1889 ordered the use of Fröhner's creolin dip officially. In this manner 750,000 sheep were treated in Prussia, in the years 1888 to 1901, and 85% were cured. Although Fröhner's method seems to have proved effective, the desirable limitation of the disease did not occur, and besides several cases of poisoning have been observed. In the year 1903 the choice of dipping fluids was left to the judgment of the attending veterinary surgeon; but the official orders recommend as effective the arsenic dips, nicotine dips, as well as the cresolic dips already mentioned. (The employment of inunctions is also permitted in mild cases.)

After the employment of bacillol baths which were heated to 35° C. according to directions, Löwel & Conze noticed rather violent symptoms of poisoning which disappeared after 2 hours; after the temperature had been reduced to 30° C. the animals were not affected.

*Arsenical Dips.* These contain as a rule a ½ to 1% solution of arsenious acid, and are generally used in France; recently, they have also been recommended again in Germany. They are very effective so that in most cases one dipping suffices, yet now and again they cause poisoning as a result of the arsenic they contain, especially if the skin suffers injury during shearing; it is for this reason that they were displaced in most countries by other dips. On this account it appears advisable to carry out the dipping only 1 to 2 weeks after shearing, and feeble or diseased sheep should not be treated at all with arsenical

dips. The udder and especially the teats and teat openings in ewes should be rubbed with fat or oil before bathing in order to prevent the entrance of the dipping fluid into the udder and subsequent diminution of milk supply. The hands and arms of operators should also be free from injuries, and the skin should be repeatedly anointed with fat or oil during the dipping process in order to save the skin from becoming brittle. All arsenical baths should only be used after the drug has been dissolved completely.

The original Tessier's dip contained 1.5 kg. of arsenic, 10 kg. of sulphate of iron, and 100 liters of water, which contents were mixed together and boiled for 10 minutes.

Clement's dip only differs from the above in containing 5 kg. of sulphate of zinc instead of iron.

Matthews' dip is iron or zinc sulphate, to which 10 kg. of alum is added. (Scheuerle & Kehm recommended a solution of 0.5 kg. of arsenic, 6 kg. of alum and 100 liters of water.)

Trasbot's dip contains 1 kg. of arsenic, 5 kg. of sulphate of zinc, 0.5 kg. of aloes and 100 liters of water; the arsenic on the one hand, the sulphate of zinc and the aloes on the other must be dissolved separately in the water and then mixed well together.

Eberhardt's dip consists of 2.5 parts of arsenic, 20 parts of alum and 300 parts of water.

In Fowler's dip 1 part of arsenic and potash are dissolved in 100 parts of water.

Viborg's dip is prepared by dissolving 1 part of arsenic and potash in 100 parts of vinegar and water.

The Prussian regulations recommend Eberhardt's, Matthews', Fowler's, as well as Viborg's dips.

The use of arsenical dips is in general quite similar to that of cresolic dips; the sheep should remain 2 to 3 minutes in the fluid and then be vigorously rubbed with brushes.

**Tobacco Dips.** The dipping fluid consists of a 2 to 5% tobacco decoction to which a definite quantity of carbolic acid or potash has been added. Tobacco dips are generally efficacious and the cost of their preparation is not high.

The tobacco dip recommended by the Prussian Government is made by boiling  $7\frac{1}{2}$  kg. of cut up homegrown tobacco in 50 liters of water for half an hour. Then the tobacco is separated by straining, 1 kg. of liquid carbolic acid and 1 kg. of potash are added to the liquid, as well as sufficient water to make up the bulk to 250 liters. The temperature of the dip should be  $30^{\circ}$  R. ( $35^{\circ}$  C), the duration of the dipping is 2 minutes. The surface of the body of sheep taken out of the bath should be worked for 2 minutes by brushing, unraveling, rubbing and milling. Manky sheep are then dipped again, and rubbed down.

Gerlach's bath is divided into 2 sections, the preparatory bath for softening the crusts consisting of 2 parts of potash, 1 part of burnt lime, and 50 parts of water, and the real scab dip consisting of 3% tobacco decoction, to be used the day after the preparatory bath. Since the tobacco decoction kills only the living mites but not the eggs, the bath should be repeated after 5 to 8 days in order to destroy the mites that have been hatched from the eggs in the meanwhile. According to Roloff it is better to use a 5% tobacco decoction, the effectiveness of the dipping being increased decidedly. A disadvantage of the method lies in the fact that the preparatory bath affects the hands of the attendants rather badly.

The dip of Kaiser & Ostertag consists in a modification of Zündel's dip, and is prepared by dissolving 1.5 kg. of crude carbolic acid, 1 kg. of burnt lime, and 3 kg. each of soda and soft soap in 260 liters of a 2% tobacco decoction.

**Other Scab Dips.** Aside from the above mentioned dips various others used to be employed which were cheaper but less reliable. As such may be mentioned:

The so-called Walz's lye which consists of 4 parts of burnt lime, 5 part of potash (or 60 parts of wood ash), 6 parts of stinking animal oil, 3 parts of tar, 200 parts of cattle urine and 800 parts of water. The lye has a very weak action,



so that in severe disease it must be repeated at intervals of 5 to 8 days, as often as 4 to 5 times; moreover, the wool is colored brown by it.

Zündel's scab dip is prepared like Kaiser & Ostertag's dip, with the difference however that pure water is used instead of tobacco decoction. In light cases one dipping suffices; in severe cases, however, a repetition of the treatment is necessary. The dip does not affect the wool (according to Zündel pure carbolic acid is to be preferred to the crude, for the oils of tar are also effective).

Recently Regenbogen has introduced akaprin baths, but Rübiger did not find these sufficiently effective (the authors noticed the same thing in sarcoptic scab of the dog and horse).

The lime and sulphur dip has recently been used more extensively in North America as well as in the English Colonies, especially in South Africa and Australia. These dips have different constituents, but are generally prepared from sulphur, lime partly slaked and partly unslaked, and boiling water (Salmon & Stiles). The results are quite favorable.

1. Recently therosot baths have now and again been employed in Germany (1 part of fluid therosot in 4 parts of water); in isolated cases, however, poisoning has been noticed, which is attributable to the mercury contained in the remedy. Especial caution seems to be necessary in the treatment of animals and in sucking ewes.

After each treatment of sheep it appears necessary to thoroughly cleanse the sheep house and disinfect the ground, the walls and racks. For this purpose the ground is thoroughly soaked with hot lye after the straw and the upper layer of dirt have been removed and before the ground is disinfected. The wooden parts and the walls are also washed with lye and are painted with fresh lime. After the last disinfection it is advisable to ventilate the stable thoroughly for two weeks before sheep are permitted to enter it.

During the treatment as well as previously to it the sheep should, if possible, be fed intensively, for they are much weakened by the energetic treatment, and those that are severely ill may be completely exhausted. After the treatment is completed the flock should be watched for some time (in Germany a period of 8 weeks is prescribed) in order to determine a definite cure; the disappearance of itching and the even aftergrowth of wool are especially to be noted.

The treatment of sarcoptic and dermatophagic scab consists in removing the softened crusts and in the repeated dressing of the affected parts of the body with anti-parasitic ointments and oily substances (see page 951).

**Literature.** Brandl & Gmeiner, W. f. Tk., 1901, 229 (Lit.).—Fröhner & Wittinger, Der preussische Kreistierarzt, 1905, II, 351.—Gerlach, A. f. Tk., 1877, III, 326.—Grün, W. f. Tk., 1905, 602.—Kaiser, Vortr. f. Tzte., 1886, IV, 10, H.—Löwel, Conze, Pr. Vb., 1908, I, 94.—Matthieu, Rec. 1856, 434.—Mathis, J. vét., 1898, 25.—Noack, S. B., 1898, 95.—Ostertag, D. t. W., 1908, 459.—Paszotta, Monh., 1901, XII, 243.—Pr. Vb., 1902, I, 145.—Rübiger, Monh., 1901, XII, 190.—Salmon & Stiles, Anim. Ind., 1897, 98.—Schlampp, Therap. Technik, 1906, I, 216.—Schleg, S. B., 1877, 83.—Walz, Natur. u. Behandl. d. Schafräude, 1812.—Zürn, W. f. Tk., 1874, 121.

#### (d) Scabies of the Goat.

Mange of the goat is most frequently caused by *sarcoptes scabiei* s. *squamiferus*; much more rarely is the disease caused by the *dermatophagus communis*, while *dermatocoptic* scab (in the external ear passage) has hitherto only been observed by Nallet, Morat and Mense.

1. **Sarcoptic Scab.** It begins on the face, especially on the lips, around the nasal openings and on the ears, passing, however, in severe cases to the body, the lower abdomen, the udder and finally to the extremities. At first gray, dry, bran-like scales form, later on, however, the skin at these places is covered with large crusts which are bluish gray, glistening, fissured and hard, and is also thickened and wrinkled; the hair falls out, and the bald spots formed in this manner coalesce to produce large hairless patches. Itching is very marked. In this severe form the nutrition of the animals suffers very much, and in consequence they die not infrequently (the mortality figure may be as high as 50%).

Scabies of goats is apparently transmitted now and then to horses, cattle and pigs (Wallraff) and in several enzootics man has also been affected (Rolloff transmitted the disease artificially to short-wool sheep with smooth fleece that does not get soaked by perspiration).

2. **Dermatocoptic Scab (ear mange).** This form of scab, which occurs enzootically in the Congo State, manifests itself by the collection of brown, fungus-like scabs in the external ear passage, which form firm plugs in the meatus and lead to deafness. The appetite is diminished and the animals perish after several months of illness.

3. **Dermatophagic Scab.** According to Delafond, this begins on the neck, along the line of the back, eventually on the root of the tail, and then spreads to the sides of the body; the face, ears, extremities, udder, and tail remain exempt. By this localization alone sarcoptic scab might be differentiated. Otherwise the skin changes are similar to those of sarcoptic scab, only that here the affection spreads more slowly and its course is milder. Parker saw no falling out of hair in one case but only the formation of hard scabs on the thickened skin, which were damp on their under surface.

**Treatment.** Since dipping is badly borne by goats (Wallraff used Walz's dip with success), treatment is limited to the application of anti-parasitic ointments and oils (see pages 942 and 951). Clipping the hair promotes the successes of treatment.

**Literature.** Hable, Ö. V.j., 1877, XLVII, 53.—Mense, *Handb. d. Tropenkrankh.*, 190—, III, 791.—Rolloff, A. f. Tk., 1877, III, 311.—Sander, *ibid.*, 1896, XXII, 63.—Wallraff, *Rep.*, 1854, 297.

#### (e) Scabies of the Dog.

True mange of the dog is caused by the *Sarcoptes scabiei* s. *squamiferus*; in addition to this parasite the *Dermatophagus communis* (var. *canis*) is found exclusively in the external ear passages.

1. **Sarcoptic Scab.** This disease, which is very general in dogs, occurs most frequently on the head, the bridge of the nose, the orbital rims or on the base of the ears; not infrequently, however, the morbid changes occur on the front of the chest, on the lower abdomen, in the axilla, on the inner surfaces of the thighs, on the root of the tail, and subsequently they pass on to the other parts of the body, and finally extend over the whole body. Little papules appear on round inflamed places, some of them changing to vesicles or even to pustules, especially on thin-skinned places. On the diseased skin profuse bran-like scales are formed at first, and later yellowish gray crusts; the skin thickens in consequence of frequent scratching or rubbing and exhibits thick folds on face, neck and chest. The hair falls out prematurely and in this way irregularly shaped bald spots form, which coalesce into large hairless patches. As in horses this form of mange frequently runs its course accompanied by profuse desquamation (Fig. 145).



Fig. 145. *Sarcoptic mange* in the dog.

The development of the disease is mostly accompanied by violent itching, whereupon the animal becomes very restless if kept warm, and injures the affected places by frequent scratching, gnawing and rubbing. If the disease is prolonged and spreads to the greater part of the body, the animals become emaciated and at times emit a peculiar mouse-like smell (Hébrant & Antoine). Finally, if no treatment is undertaken, they die with symptoms of cachexia.

2. **Dermatophagic Scab.** (Ear mange. Scabies auricularis.) In the external auditory meatus of the dog dermato-



phages frequently occur. Thus Becker found dermatophages in the external auditory meatus in 17.3% of all dogs suffering from external otitis, while in dogs without ear disease none were found. Otherwise the mites seem to occasion only itching and do not of themselves cause any inflammation, the skin of the ear being found uninjured now and then in spite of an abundant invasion. The otitis, which is frequent when mites are present, is a secondary affection, being due to the fact that injuries and soiling of the outer ear and auditory passage easily arise from scratching and from shaking the head (see page 854). The symptoms which may be referred to the presence of dermatophagus mites consist in frequent scratching of the ears, shaking the head, whining, howling and rubbing the ears on any convenient objects (Becker). These signs of itching are especially noticed in warm places and during the night. Subsequently baldness of the lobes of the ears, scratches, abrasions, bloody ears, and finally the symptoms of otitis externa become manifest (see page 854). In addition epileptiform spasms and deafness have also been reported (Méglin, Nocard, Becker). In the absence of inflammatory products in the ear passages the mites may be seen with the unaided eye as fine, grayish white and partly movable points, or they cover the surface of the skin with an adherent powdery layer (Becker). They are most numerous near the drum of the ear.

**Diagnosis.** Sarcoptic mange may be mistaken for acariasis, and all the more so as this very frequently commences on the above mentioned parts of the head. But here itching is quite slight or completely absent, and one often sees bluish red pustules on the skin which always contain many acarid mites. Sarcoptes mites are on the contrary difficult to demonstrate. Both these forms of scab may be present at the same time in one animal. The difficulty in obtaining mites makes it hard to distinguish scab from dry eczema, in case this is not limited exclusively to the back but occurs also on other parts of the body. Intense itching which is out of proportion to the changes in the skin is indicative of scab. Other parasites (lice, fleas, ticks) may be seen with the naked eye.

**Treatment and Prophylaxis.** To secure lasting results it is very advisable to extend the treatment over the whole body. It should be commenced by clipping the hair at least over the diseased places and round about them, after which the parts of the body that are covered with crusts may be rubbed with green soap, the softened crusts being removed with brushes and lukewarm water after 1 to 2 hours. The treatment proper then follows on the next day and is most appropriately carried out in sections; thus each day only a third or a fourth of the surface of the skin is treated energetically, the dressing being left on the skin for 4 or 5 days and then washed off with luke-

warm soap and water or with  $\frac{1}{2}$  to 1% liver of sulphur solution. This operation must be repeated, with intermissions of 3 to 4 days until the skin forms no further crusts and the itching has disappeared completely. One must restrain the animal as much as possible from licking the application (muzzle, binding the mouth, broad collar of stiff paper, etc.).

Of the remedies which are capable of killing the mites, the following are the most useful: tar in the form of liniment (pix liquida, sapo viridis aa 1 part, alcohol 1 to 5 parts); creolin, lysol, and ichthyol in similar form and composition; creosote (in 10% oily mixture or with 5 parts of green soap and alcohol); further for sensitive dogs and for the region of the eye and nose, Peruvian balsam and styrax may be used (pure or mixed with some alcohol, glycerine or oil). According to Fettick, eudermol (salicylate of nicotine) in the form of a 1% ointment has answered well (expensive!). Cresol liniment (aqua cresolica 2 parts, soft soap and alcohol aa 1 part) is of excellent service in mild cases. Winter found a watery solution of therosot (1:4) rapid and safe in its effect. When employing tar or carbolic preparations it is advisable to give Glauber salts internally to the affected animals (2.5 gm. per day) in order to avoid phenol poisoning, or at least to watch the general condition and the urine for any signs of commencing intoxication with phenol.

For the treatment of ear scab the external auditory meatus is first to be cleansed, after which carbolic acid or creosote in oil or glycerine (10%), also naphthol oil (naphthol 10.0 g., ether 30.0 g., olive oil, 100.0 g.), may be instilled for a few days.

During the treatment the animals should be fed well and protected from cold. As prophylactic treatment careful disinfection of the stable and of blankets, etc., is recommended, and the diseased animals should be separated from other animals until their recovery is complete.

**Literature.** Albrecht, W. f. Tk., 1901, 194.—Becker, Monh., 1907, XVIII, 547 (Lit.).—Brandl & Gmeiner, W. f. Tk., 1900, 177.—Deich, S. B., 1903, 84.—Fettick, Z. f. Tm., 1901, V, 291.—Fröhner, A. f. Tk., 1887, XIII, 341.—Méglin, Rec., 1881, 129.—Rievel, D. t. W., 1901, 105.—Schlampp, Therap. Technik, 1906, I, 234.

#### (f) Scabies of the Cat.

**Sarcoptic Scab.** Mange caused by the *Sarcoptes minor* begins on the ears and in their immediate neighborhood, then it generally passes on to the head, but exceptionally also attacks the feet and the sacral region.

In conjunction with intense itching the animals wipe the head with their paws, shake the head and rub on neighboring objects. Small papules and vesicles form on the skin, and soon this is covered with a thick, bran-like layer which later on gradually changes to rather thick, grayish yellow, dry, fissured scabs



(Fig. 146). Hand in hand with this process the hair falls out on the diseased parts, while the skin itself becomes thickened and wrinkled. In severe cases the eyelids swell and purulent conjunctival catarrh sets in. Owing to swelling of the alæ of the nostrils breathing becomes difficult, the animals become emaciated and die of the disease in 3 to 6 months.

**Dermatophagic Scab.** (Ear mange.) This manifests itself by similar symptoms to those in the dog (see page 956) but occurs very seldom.

**Diagnosis.** The localization of the skin disease leading to the formation of crusts in the neighborhood of the ears and on the head is characteristic of the affection; the microscopical examination of the crusts, in which the mites are present in great quantity, makes the disease easily recognizable.

#### **Treatment.**

Since cats will not stand washing and bathing, the treatment is limited to the use of ointments which can be removed by rubbing with dry bran. The best ointment is that of Helmerich



Fig. 146. *Sarcoptic mange* in the cat.

(sulphur sublim. 150 gm., potassium carb., 8.0 gm., adeps suilli 60.0 gm.). The treatment of small surfaces with Peruvian balsam has afforded good results, but this remedy may cause severe symptoms of brain irritation, and even death, especially in an extensive spread of the disease (Schindelka, Fröhner). Styra (with 4-5 parts of alcohol) can also be used and is less dangerous. Schindelka always found sulphur in oil effective. Carbolic acid and tar preparations are to be avoided on account of their poisonous effects on cats. Mange of the cat is transmissible to man, horses, cattle and dogs; one must therefore take care to isolate the affected animals.

The ear mange necessitates cleansing of the ear passage with Peruvian balsam and glycerine, aa, or naphthol oil.

**Literature.** Dinter, S. B., 1862, 99.—Köhne, Mag., 1868, 288.—Méglin, Rec., 1863, 68.—Schwartz, Rep., 1875, 165.



**Scabies in Wild Felines.** In zoological gardens lions and leopards may become affected with scab due to *Sarcoptes communis* (Johne, Koolisch, Delafond & Bourguignon), manifesting itself by intense itching and the occurrence of spots covered with crusts on the trunk, whence the disease extends quickly over the whole body. The nutrition of the animals is much interfered with and some of them die.

The treatment consisted, in John's cases, in syringing with a spray of Peruvian balsam (1:3 parts of alcohol) and afterwards rubbing with long handled brushes. Five repetitions at intervals of 8 to 10 days effected a cure. (Johne, A. f. Tk., 1880, VI, 146.)

Among other predatory animals, foxes are not infrequently affected with sarcoptic scab and sometimes also infect hounds. (Deich, S. B., 1903, 84.)

### (g) Scabies of the Pig.

**Sarcoptic Mange.** This form of mange is caused by the *Sarcoptes scabiei* v. *squamiferus* and occurs very frequently, and as an enzootic, especially in large piggeries. Marked and dangerous symptoms are seen especially in swine with a thick curly hair growth, while now and again it has been noticed that pigs of the English breeds living with these have not been affected. It may here be stated that scab is very frequent in pigs of the Hungarian breeds. Young swine or sucklings are affected most often. Insufficient nourishment, debilitating diseases (hog cholera, pyobacillosis, rickets, etc.) and other complaints, and even inoculations for erysipelas may be followed by mange, previously apparently healthy swine being affected in great numbers.

The disease develops with violent itching, especially on the head and in the neighborhood of the eyes and ears, on the back, on the sides of the body and inner surfaces of the thighs (Fig. 147). Dry, bran-like scales collect upon the small itching nodules, the bristles fall out and large crusts form which are said to be grayish white, so that the animal appears as if sprinkled with dry guano. The authors' own numerous observations have led them to conclude that the scabs are mostly brownish black on account of the admixture of particles of dirt. The skin thickens in course of time, and is found in thick, strong folds (Fig. 147), between which superficial ulceration and even abscess formation may occur.

The disease develops slowly, and in its severe form it hinders the development of the animals or their fattening. Now and then it leads also to cachexia and causes numerous deaths.

Johne saw sarcoptic scab in swine occurring as an enzootic; on account of the wrinkling of the skin it proved to be incurable.

The mange mites of swine live also on the skin of other animals as well as of man and here cause a local eczematous disease which generally heals spontaneously in a few days.

**Diagnosis.** From eczema or the so-called "smut" of young pigs or from pitch scab, this true scabies is differentiated by the very intense itching; but with certainty only by the discovery of the mites, which is usually easy. The mange of pigs is also mistaken very often for so-called "smut" of young pigs which doubtless occurs rarely as a non-parasitic eczema (Sohnle, Albrecht, Mayer, authors' observation).

**Treatment.** This consists in removing the scales and crusts by brushing with soap and water or by scraping with a wooden spatula, and afterwards using parasiticides. In isolated cases of the disease the following may be used: Helmerich's ointment; an intimate mixture of oil of turpentine and flowers of sulphur (1:8); Fritscher's ointment (flowers of sulphur and ung. hydr. cin.  $\bar{a}\bar{a}$  14 parts, ol. anim. fetid 7 parts,



Fig. 147. *Sarcoptic scab* in the pig.

ol. cannabis 84 parts), or Imminger's ointment (hydrarg. metall. and sulphur. flores  $\bar{a}\bar{a}$  5 parts, canth. exacte pulv. 2 parts and vaseline 40 parts); cresol liniment, according to Brandl & Gmeiner (Liqu. cresoli sap. 5 parts, soft soap 10 parts, water 85 parts). These applications may be rubbed alternately into one-half of the body. In large outbreaks mange baths are more suitable and may be carried out (see page 952) even during the cold season, in warm stables. Since adult swine may carry the mites in infected piggeries although not affected at all or at most only slightly, the treatment should be extended to them likewise.

For the rest the same prophylactic treatment should be adopted as in sheep scab (see page 954).

**Literature.** Albrecht, W. f. Tk., 1901, 58.—Brandl & Gmeiner, *ibid.*, 1900, 489 (Lit.).—Marek, Z. f. Tm., 1904, VIII, 288.—Mayer, W. f. Tk., 1905, 709.—Sohnle, Rep., 1891, 74.

face of the shell of the ear (Fig. 148). At times the orifice of the ear, as well as the middle ear or the petrous bone is affected with a violent inflammation (Zürn, authors' observation) and the head is then held obliquely. Exceptionally the inflammation extends to the coverings of the brain. After the development of these complications the animals die. (Zürn and Schindelka also saw sarcoptic scab on the skin of the bridge of the nose.)

Dermatophagus mites occur only exceptionally in the external ear passage of the rabbit, and then together with dermatocoptes mites.

**Treatment.** For this the following may be employed: Helmerich's ointment, formalin paste; according to Gmeiner especially oleum carvi (5% ointment). Reichert made the observation that a third of the body may be treated at one time with 12 gm. of cresol liniment (amount for 1 kilogram of body weight) without injurious effects. On the other hand, washings or baths are very badly borne by rabbits. Ear mange may be treated with 5 to 10% creolin or carbolic oil or by painting with glycerinated creolin or carbolic acid; according to Gmeiner here also oleum carvi (mixed with 10 parts of almond oil) answers best.

**Literature.** Gmeiner, D. t. W., 1903, 69 (Lit.); A. f. Tk., 1906, XXXII, 170 (Lit.).—Neumann, Rev. vét., 1892, 141.—Zürn, W. f. Tk., 1874, 277.

### (i) Scabies of Fowls.

1. **Foot Mange.** (Foot itch; Gale des pattes [French].) This form of scab is caused by the *Knemidocoptes mutans*, which mite was formerly generally known as *Sarcoptes* or *Dermatoryctes mutans*.

The *Knemidocoptes mutans* (Fig. 149) is a little animal that is very similar to the *sarcoptes* mite, having, when fully developed, four stubby pairs of feet (the clinging disks are rudimentary in the female) and a short conical head; immediately behind the head, on the back, there is a U-shaped chitinous structure, and the dorsal part of the abdomen shows scaly formation. The male is oval, 0.2 to 0.25 cm. long and 0.15 to 0.17 mm. wide, with two long bristles at the hind end; the female more rounded, 0.4 to 0.45 mm. long, 0.35 to 0.38 mm. wide. The mite bores passages into the epidermis.

Brandl & Gmeiner and Reichert found that of all remedies investigated against the *Knemidocoptes mutans*, liquor cresoli saponatus in watery solution had the strongest effect; also chloroform (pure or with 10% of oil), further creolin, bacillol, lysol killed the mites very quickly, as did also etheric oil and Peruvian balsam with oil or alcohol.

The skin disease caused by this mite occurs mostly in blooded fowls (Cochin China, Brahma, Bantam, Dorking), less often in the native breeds, exceptionally in turkeys, pheasants, pigeons and singing birds. Ostertag observed the transmission of the mites to a horse. The development and spread of scab is promoted by crowding of the fowls, although even under these



conditions the extension is only slow and some birds of a flock always remain healthy. The affection attacks exclusively the legs downwards from the tarsal joint. Little grayish white scales appear mostly on the anterior surface of the tarsus and toes, which afterwards spread by continuity and gradually change into gray scabs 1 cm. thick, rough and yellowish (Fig.



Fig. 149. *Knemidocoptes mutans*. Female, on the left viewed from the back, on the right from the abdominal side, in the middle an egg. Magnif. 75. (After Mègnin.)

150). These show a leaf-like construction. Some lamellæ have a peculiar lardaceous or mother-of-pearl luster, and adhere to the surface of the skin. The foot, which is covered with crusts down to the toes, has the appearance of having been plastered thickly with lime or mortar (so-called lime leg, scaly leg, elephant limb, fish scale disease). Between the lower lamellæ of the scabs numerous

mites are to be found in different stages of development, while the skin appears inflamed and raw. Itching is manifested by the restlessness of the animals and by pecking into the scabs with the beaks.

Bending of the joint and motion are interfered with by the armor-like scabs which surround the foot. The birds become lame; in consequence they are soon unable to stand and squat down continuously. In many cases arthritis sets in as a complication, and single toes or even all the toes may be lost. In such severe cases the birds fall off in condition and finally die of cachexia. Several months or even a year may elapse, however, before the disease reaches this degree.

The involvement of the head and neck mentioned by many authors in connection with foot mange was undoubtedly caused by the *Knemidocoptes laevis*.

The treatment consists in softening the scabs with glycerine or soft soap, rubbing with a little brush and subsequent applications to the dried diseased surfaces with any anti-parasitic remedies, of which Helmerich's ointment is perhaps the best. Of other remedies Peruvian balsam and styrax may be employed, while tar or carbolic preparations appear little suited for young animals. Brandl & Gmeiner found cresol liniment (cresol water 5 parts, soft soap and alcohol  $\bar{a}\bar{a}$  2.5 parts) or cresol ointment (1 part of cresol, 10 parts of paraffin ointment) very effective. Reichert recommends the use of oleum carvi in ointment form (1:5). Apart from very neglected cases, a cure generally results.

To insure success disinfection of the fowl houses and perches is necessary.

2. **Mange of Feathered Parts of the Body.** (Scabies *deplumans*.) This form of scab is caused by the *Knemidocoptes* (*Sarcoptes*) *lævis*.

The *Knemidocoptes lævis* which has been called *Sarcoptes lævis* by Railliet is very similar to the *Knemidocoptes mutans*, from which it differs in that the female possesses no shield-like chitinous formations on the back, the U-shaped chitinous structure is less developed, the body more circular and the hind pair of legs project over the edge of the body. The differences in sizes between these two kinds of mites are inconstant, as the authors had an opportunity of ascertaining.

The *Knemidocoptes lævis columbæ*, the *Kn. lævis gallinæ*, *Kn. lævis phasiani* are only subdivisions of the *Knemidocoptes lævis*.



Fig. 150. *Foot mange* in the fowl.

This form of mange occurs in chickens, sometimes as an enzootic, also in pigeons and exceptionally in pheasants. It occurs particularly in spring and summer, and disappears almost completely with the approach of autumn, but reappears the following spring. It usually begins in the sacral region and then passes on to the back, the abdomen, the thighs, the neck and the head. Frequently, however, the head or the upper part of the neck may be first attacked (Fig. 151). Contagion seems to spread chiefly by treading, consequently the loin re-



gion is the first to be attacked. On the affected parts of the body, falling out of the feathers is noticeable, often also they break off at the level of or close to the surface of the skin; the large feathers of the tail and wings are not involved. The stumps of feathers remaining in the feathered bulbs become cleft and fine scales form on the skin. At the periphery of the bald patch the quills of drawn out feathers are covered with epidermis scales in which the mites are demonstrable. The skin itself is elastic, pale red and not noticeably thickened; at times, however, one finds it inflamed and dotted with nodular formations. In such cases the hens lay fewer eggs, emaciation sets in, and



Fig. 151. Scab caused by *Knemidocoptes levis* on the neck and head of a cock, with favus on the comb.

now and then the birds become cachectic and die. Cocks are attacked more severely as a rule.

The falling out of the feathers due to *Knemidocoptes levis* has, according to Neumann, frequently been mistaken for abnormal moulting. According to the same author most cases of feather picking are caused by the *Knemidocoptes levis*.

Mange of the feathered parts of the body occurs now and then in conjunction with foot mange, and leads in such cases to the supposition that scab of the feathered area may be due to *Knemidocoptes mutans*. A mistake is quite easily possible



since both varieties of mites present only few differences (see page 965).

In geese, Railliet observed scab of the feathered parts of the body caused by the *Knemidocoptes prolificus*. On the head, especially around the beak and the eyes, further under the mandible, punctiform, hard nodules formed which contained sexually mature females, also many eggs and larvæ of the mites.

Railliet was successful in treating scab of the feathered parts with daily sulphur baths; fresh feathers grew on the bald surfaces. In general the same remedies may be employed as in foot mange.

**3. Dermatophagic Scab.** It is caused by the *Dermatophagus gallinarum* (*Epidermoptes bilobatus* Rivolta and *E. bifurcatus*) and begins on different parts of the body, principally on the neck and breast; in severe cases it may even attack the whole body, the comb and the wattles not excepted (Friedberger). Fine, transparent, yellowish scales first appear on the skin, mostly without itching (Rehm also noticed itching) which thicken later on to form strong, dirty brown, doughy crusts. Then the skin appears thickened and tumefied (Rehm). Within a short time the birds become emaciated and exhausted, and not infrequently a fatal termination is observed. This form of mange now and again develops along with foot mange and perhaps also with scab of the feathered parts of the body.

Since dermatophagus mites not infrequently produce no disturbances in the health of fowls, one should always make sure when they are present whether in such cases the skin changes may not be brought about by vegetable parasites (*Tricophyton*, *Achorion*) (Neumann).

The treatment is the same as in foot mange. Kitt recommends a 5% solution of epicarin.

**Literature.** Brandl & Gmeiner, W. f. Tk., 1900, 349 (Lit.).—Friedberger, D. Z. f. Tm., 1881, VII, 281.—Méguin, Les parasites et les maladies parasitaires, 1880.—Neumann, Maladies paras. des oiseaux dom., Paris, 1909.—Rehm, S. B., 1901, 264.—Reichert, Die Fussräude d. Geflügels, Diss. Giessen, 1909 (Lit.).

## 22. *Acarus Mange. Acariasis.*

(*Akarusausschlag*, *Haarsackmilbenausschlag* [German]; *Gale domestique* [French].)

The acarus mange of the domestic animals is a contagious skin disease caused by the acarus mite, which is characterized by the absence of, or by slight itching, by falling out of the hair mostly with simultaneous desquamation, or by pustule formation with subsequent decided thickening of the skin.

**History.** The acarus mite was discovered by Berger (1841) in the external ear passages of man, but he did not announce his discovery

until 1845, attention having been called a month after his finding to the occurrence of mites in the comedones of man by Heale (1841), and soon after by Simon (1842). In the dog the hair follicle mite was first found by Tulk (1844); in the horse by Wilson (1844), and Gros (1845); in the cat by Leydig (1859); in the pig by Obermeier and also by Korzil (1878); in cattle by Faxon (1878); in goats by Niederhaeusern (1881); and in rabbits by Pfeiffer (1903). Besides these authors, Miescher (1843), Gruby (1845), Mégnin (1877-1892), Csokor (1879) and recently Gmeiner (1908) have investigated the hair follicle mite; the last mentioned author also elucidated the pathogenesis of the complaint.

**Occurrence.** Acariasis is a frequent and obstinate disease of dogs; it appears to be prevalent all over the world and, according to Lemke, has hitherto been found to be absent only in Eskimo dogs. Exceptionally it also occurs in cats; pigs are attacked somewhat more frequently, but on the whole rarely and only in certain neighborhoods, but then they fall sick in great numbers. Isolated cases have also been noticed in cattle, goats, horses and rabbits, while in sheep these mites cause no skin changes (Oschatz found acarus mites in the Meibomian glands of sheep). Acariasis has further been noticed in a Sambu stag, in a roebuck, also in field mice, in house mice, bats and rats. In so-called grubs (comedo) of man the mites are also encountered frequently, but generally occasion no special injury.

Fröhner found 2 per cent of acariasis among 70,000 dogs treated in the Berlin clinic. At the Budapest clinic 2% of the dogs attended proved to be affected. In the Vienna clinic 10% of all dogs with skin disease are affected with acariasis.

According to Stiles acariasis is not rare in cattle in North America, especially in the southern districts where very considerable damage is caused, since only parts of the skin of the attacked animals is suitable for tanning.

In pigs Csokor and Legram & Régulato saw an epizootic occurrence of the disease.

**Etiology.** The hair follicle mite (bulb mite, *Acarus s. Demodex folliculorum*) belongs to the order of Acarina, of the



Fig. 152. *Acarus folliculorum*. Magnif. 150.  
(After Mégnin.)

class of Arachnoidea as representative of the family Dermatophila (Demodicida). Its body is elongated, lancet-shaped, or like a laurel leaf; at the thorax the three-jointed legs are given off immediately behind one another, on both sides (in the larva three and in the fully developed animal four pairs), while these are absent on the long, conical, cross-striped abdomen, which is serrated at the edges. The lyre or horseshoe-shaped head is provided with a movable rostrum, with upper and lower jaw and also with feelers (Fig. 152). The male is smaller than the female; the eggs are oval spindle-shaped, the larvæ hatched from the eggs are similar to fully developed mites, but have only three rudimentary pairs of legs and attain their full development after three moultings.

In the domestic animals the following hair follicle mites occur:

1. *Demodex folliculorum* (var. *canis*); the head and chest part somewhat shorter than the abdomen; the male 0.22 to 0.25 mm., the female 0.25 to 0.30 mm. long; both 0.045 mm. wide; the eggs spindle-shaped 70 to 90 microns long, 25 microns wide. Originator of acariasis in the dog.

2. *Demodex phylloides*; comparatively broad, laurel-leaf like, the length of the head and chest part and of the post-abdomen about equal; the male 0.22 mm. long, 0.057 mm. broad, the female 0.25 to 0.26 mm. long, 0.06 mm. wide; the eggs oval, constricted at each end (Csokor). Cause of acariasis in the pig.

Besides these one encounters special varieties of mites in the sebaceous glands of the other animals.

Of the tenacity of hair sac mites the following may be stated from the investigations of Brandl & Gmeiner and those of Gmeiner:—In dry air the mites are capable of living  $1\frac{1}{2}$  days at most, while in very moist atmosphere they remain alive as long as three days, but they are killed in a few seconds by a temperature of over  $41^{\circ}$ . They resist the cold of winter for three days at most if the humidity is high.

Of chemical substances the hair follicle mites are killed immediately by acid. carbol. liquefactum, bacillol, creolin, cresolum crudum, creosote, liquor cresoli saponatus, lysol, tar, chloroform, bisulphide of carbon; in about a minute by the ethereal oils, especially ol. anethi and ol. carvi, further aqua cresolica, creolin, cresol or tar liniment, 2 to 10% carbolic acid solution; in 2 minutes by tincture of iodine, formaldehyde-solution; in 3 minutes by salicylic acid in 10% alcohol, and 1% alcohol eudermol solution; in 6 minutes by Gmeiner's solution of ol. carvi with alcohol and castor oil. The mites were killed by Peruvian balsam (pure) in 12, by liquid styrax in 55 and by 1% corrosive sublimate solution in 19 minutes.

The **infection** occurs undoubtedly in the great majority of cases by immediate contact of healthy with diseased animals. Short-haired and young dogs are especially susceptible (among 177 dogs observed by Schindelka to be suffering from acariasis, 141 were short-haired, and only 36 long-haired), which is accounted for by the fact that in short-haired dogs the mites gain access to the skin more easily, and in young animals they penetrate more easily into the orifices of the hair follicles. Certain skin diseases (eczema, sarcoptic scab) as well as distemper also increase the susceptibility to a certain degree. In contrast to the frequency of the eruption, the artificial transmission of the disease to healthy animals has succeeded only rarely, and at most a transitory skin affection occurred after rubbing in pus containing the mites (Haubner, Cornevin); only Guinard succeeded in producing the disease in its typical form in a young dog. It is not a rare occurrence that some dogs of the same breed and kept under like conditions are not infected by their companions. For the infection to occur, certain predisposing circumstances not yet known are evidently of influence. (Prietsch observed contagion of a billy goat and a deer from a dog.)

**Pathogenesis.** The hair follicle mites penetrate through the orifice of the hair follicle, and subsequently also into the regional sebaceous glands; they increase there so that their number in a hair follicle or sebaceous gland may amount to from 30 to 60 (Friedberger) and even 100 to 200 (Gruby). They occur mostly in all parts of the hair follicle and sebaceous gland and lie almost



In dogs in which acariasis is of the greatest practical importance on account of its frequency and severe course, the illness appears in two principal forms.

The *squamous form* declares itself as a squamous eczema which remains limited for a time to small surfaces. On the orbits and in their neighborhood, further on the elbows or on single toes, more rarely on the body, the hair falls out in small patches, and in this way rounded, often sharply defined pronounced bald spots (Fig. 154) arise, reminding one of alopecia areata. At these places the unpigmented skin is somewhat reddened and mostly covered with bran-like scales. Sometimes it is only bald and may contain small nodules. After a certain time the skin assumes a bluish gray color on the affected spots. In many cases the affection begins with round, red or copper-colored spots and scarcely perceptible scaling. But after a time these spots assume a lead gray color (Schindelka). Itching is absent or at most slight.



Fig. 154. *Acarus mange* in the dog. Falling out of the hair in sharply circumscribed patches.

In this form the affection may last for months, and it may even remain unchanged to the end; it causes scarcely any itching and exerts no influence on the general condition of the affected animal. But often the pathological changes in the skin attain a high degree, or the affection occurs from the beginning in the following described form.

In the *pustular form* (Fig. 155) hemp nodules, which subsequently change to pustules swollen and inflamed skin of the regions already. Then the neck, the chest and the inner surface of other places are attacked. These often appear red, and reddish pus or a tallow-like thick numerous mites in different stages of development from them by pressure. The skin is arranged between which it is intensely inflamed and appears many brown or more gray scabs, as well as with The hair falls out over the whole area that scales or over the greater part of it (Fig. 155).

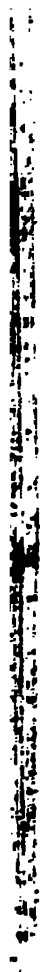


Fig. 155. *Acariasis* in the dog with pustule formation and thickening.

In this form also the itching is slight, or completely absent. The surface of the unpigmented skin appears pinkish red (so-called red mange), the skin of the head and ears has a brown or warthog color. Through the presence of numerous pustules and folds of skin it presents the appearance of bark (Fig. 156). If in such cases intense itching and suspicion is aroused that sarcoptic scab is also present (delka). In this case the frequent scratching and increase the inflammatory process and ulcers may develop of the broken pustules, through which cavities in the cutaneous connective tissue may be reached. At







the affected parts of the body swell visibly. Then the animal falls away greatly in condition, gives off a very objectionable smell, and finally dies, completely exhausted, as a result of intoxication or general septic infection.

In many cases the disease extends over the whole body, the neck appears covered with bran-like scales, and scattered single pustules may be present, yet the hair falls out only in isolated circumscribed places and the skin here shows the appearance of moist eczema.

Horneck found the mites in the secretions from the eyes, ear and prepuce of dogs affected with acariasis.

In **cats** the hair follicle mites very rarely cause disturbances. The head, round about the eyes, nose and ears are spots of predilection of the disease, which causes similar changes as in the dog, although at times they are transitory and not very pronounced. Sometimes the hair follicle mites are encountered in the external ear passage without having caused any symptoms of disease (Hyrtl, Mégnin).

In **pigs** the disease assumes a similar form to that in the dog, only that here the mites live in the sebaceous glands, and besides purulent vesicles are formed through coalescence of pustules, which are as large as a hazelnut and may contain 1,000 mites; after breaking up they may lead to ulceration without any tendency to healing. Now and then, however, only millet-sized to lentil-sized com-

edo-like nodules appear in the skin, from which a soft pulpy mass can be expressed (Knoll). These changes are generally found on thin-skinned spots (around the snout, cheeks, forehead, lateral surfaces of the neck and body, lower belly, in the folds of the stifle, inner surfaces of the thighs), while the back of the neck, the back and the external surface of the thighs almost always remain healthy, even in severe cases. According to Rieck, isolated pustules not infrequently occur in the folds of the stifle. In a case described by Müller the skin of the legs of a pig had the appearance of pearl beading.

In European **cattle** acariasis is very rare, but may, in large herds, attain the form of an enzootic, as the observation of



Fig. 156. *Acariasis in the dog.* Folds and thickening of the skin on the head and fore legs.

Bügge proves, who encountered the disease most frequently in 4 to 8-year-old animals, but sometimes also in younger animals and only very rarely in older ones. With the exception of a case in which Gros saw the disease on the muzzle of a cow, the skin affection usually prefers the body, neck and shoulders (Öhl, Grimm, Bügge); it may, however, affect the whole surface of the body (Büchli). In Bügge's cases, in which many animals were affected simultaneously, the hock joint was the central point of the disease. On the skin one finds nodules of hazelnut size, conical and closely crowded, hairless, and at times reddish colored, from which purulent, cheesy masses with very many mites may be expressed. Bügge also noticed moderate itching, and in consequence the animals licked the diseased places. While Öhl sometimes saw spontaneous healing, in other cases cure did not always result even after suitable treatment.

In the **goat** the hair sac mites occasion only quite exceptionally pea to hazelnut-sized reddish nodules, occurring principally either on the body (Niederhäusern, Nocard, Railliet), or on the head and legs (Bach). Slight itching may also be present. Healing does not result even after appropriate treatment.

In the **horse** Gros saw in one case reddening of the skin around the nose, while Walther noticed in a horse intense itching on the left side of the root of the tail, and hairless, round spots as large as a twenty-five-cent piece on which the swollen skin was covered with sticky fluid oozing out like drops of sweat. In Schenzles' cases the disease simulated, on the other hand, alopecia areata, and started on the head at the bridge of the nose, around the eyes, on the forehead, nape of the neck and in the region of the parotid gland, with the occurrence of hairless spots as large as a fifty-cent piece, irregular in form and with poorly defined boundaries. Subsequently the circumscribed loss of hair extended backwards to the shoulder region, and finally spread over the whole body, after repeated rubbings with carbolized glycerine. At this time fine scales appeared on the otherwise healthy skin. Itching was absent up to the end of the attack.

In **rabbits**, Pfeiffer observed acariasis in China. The affection began with falling out of hair and scaling around the eyes, whence it extended to the root of the ear and the external and internal surfaces of the shells of the ear as well as on to the skull. In the meantime it caused folds and thickening of the skin, scabs, copious secretion of purulent material, as well as destruction of eyelids and ears, or it produced a violent inflammation of the middle and internal ear, and now and then occasioned even a fatal inflammation of the membranes of the brain; the eyeballs remained uninjured except for a superficial keratitis. The acarid mites found in the contents of the hair follicles were much smaller than those of the dog.

**Diagnosis.** The pustular form of acariasis produces a rather characteristic form of the disease in dogs. The often



peculiar copper-red color appears especially important diagnostically, further the thickening of the skin, together with a marked diminution in elasticity, also the pustules situated in the depths of the skin, mostly bluish red in color, and itching which is at most only slight. All these signs enable one to differentiate the disease easily from primary acne, sarcoptic scab, distemper, exanthema and from simple eczema. In the other species of animals relatively large, hairless and reddish nodules with thick contents awaken a suspicion of acariasis, yet the disease may be recognized positively only by the microscopic demonstration of the mites.

The squamous form is far more difficult to differentiate from other affections. Apart from the localization of the disease or its appearance at the same time on several parts of the body, the best conclusion can be formed by the microscopic examination of the fluid substance extruded by lateral pressure on the raised folds of skin, or of the skin scrapings. (Since in such cases the mites are present in the hair follicles only sparingly, a single test is not always sufficient.) In this form of acariasis one must exclude especially circinate ringworm, favus, eczema, such as that caused on the eyelids through conjunctival catarrh (distemper), also sarcoptic scab and alopecia. But every falling out of the hair in the dog without traceable cause must appear suspicious in this connection. As regards the differential diagnosis between sarcoptic scab and acariasis one should further bear in mind that both skin affections can be present simultaneously in the same dog.

**Prognosis.** In the large animals a spontaneous cure of the disease appears to be possible, also at times in dogs when the attack is slight; Uebele has repeatedly seen spontaneous healing in spite of extensive acariasis, and this is said to be the rule in the diffuse affection in young terriers, which is called distemper rash by breeders. On the other hand permanent healing can, in case of extensive eruption, be secured only rarely, even by proper treatment, and sometimes even when the disease is not very extensive, because the mites live deep under the surface of the skin and therefore it is difficult to destroy them with parasitocides, and besides secondary infection with staphylococci has often already occurred. As a matter of fact, not infrequently dogs that are apparently cured suffer renewed attacks after weeks or months. At times, however, complete cure or a marked improvement is noticed without any treatment.

In pustular acariasis of carnivora the prognosis is unfavorable; it therefore appears most expedient to kill such animals. A much more favorable opinion may be given in the squamous form where the complaint is limited to a small area, yet even here one should always reckon with the possibility of a further extension, and therefore be cautious in giving a prognosis, all the more so as even quite trivial and apparently slight



cases, and still more those that are further advanced, require treatment continued for weeks or months. Other things being equal, the prognosis is always more unfavorable in young debilitated dogs than in older ones. According to Schindelka, many more dogs may be cured by exercising sufficient patience and perseverance than is usually assumed (of 177 cases 50 were cured).

**Treatment.** First of all the hair should be clipped and, according to the extension of the disease, this should be done either over the diseased areas and their neighborhood, or preferably over the whole body; then, and also later, it appears absolutely necessary to split all nodules or pustules with a pointed knife and to remove their contents. In this way one gets rid of many mites, especially if a bath in a 5% solution of liver of sulphur is administered each time. The pressing out of fresh pustules and subsequent bathing is advisable also in the treatment of the later stages, and so is the employment of periodical fomentations and rubbings with soap liniment, salicylated alcohol or salicylic oil, 10% lysol vasoliniment, in order to cause loosening of the skin and detachment of the horny plugs in the hair follicles.

Of the parasitocides the most suitable of all are non-irritant substances, since by their use there is the least danger of causing an extension of the disease or of the squamous eruption becoming pustular; particularly the employment of tar cresol preparations and corrosive sublimate easily occasion a spread of the process. The remedies should be applied without force, but nevertheless thoroughly, with a soft brush, with the hand or a pad of cotton wool. Very frequent washings may also be harmful. The selection of remedies is influenced by the species of animal and by the form and extension of the disease, and also by the condition of the patient. During long continued treatment a change of the anti-parasitic remedies not infrequently becomes necessary.

Peruvian balsam is well adapted for mild cases and produces favorable effects, especially if mixed with alcohol. Siedamgrotzky produced a cure with this preparation in a severe case, but others saw no particularly good results in such cases in spite of long continued treatment. The high price of the drug limits its employment to mild cases of disease; on the other hand it can scarcely be dispensed with in acariasis localized on the head. In circumscribed disease eudermol is of good service (Fettick) in the form of 1% ointment, but it is also expensive; in many cases a brief depression becomes manifest after the application. Salicylated oil (1 part salicylic acid dissolved in 30 to 40 parts of warm oil), creolin or ichthyol (with alcohol aa), corrosive sublimate ointment (1:100) or carbolic ointment (1:10) at times give good results. The application of these remedies must be repeated several times at intervals

of a few days (see page 957). Of good service in the squamous form is caraway oil, which is recommended by Gmeiner (ol. carvi and alcohol āā 10 gm. ol. ricini 150 gm.) with which the diseased parts should be rubbed energetically once or twice daily with the fingers; a sulphur bath is given once every week. According to Brandl & Gmeiner, liquor cresoli saponatus is the most effective remedy, but the authors cannot confirm this.

Schindelka obtained healing repeatedly with bisulphide of carbon (sulfuretum carbonicum s. carboneum sulfuretum). After the skin is prepared properly, pads of bisulphide are pressed upon the affected spots for several minutes, and then a paste of formalin (formaldehyde 1 to 3, vaseline 50, zinc oxide and powdered starch āā 25) is applied to the carefully dried skin. This treatment repeated three to five times at intervals of 3 to 4 days is said to accomplish its object as a rule. In cases where the skin is much thickened Schindelka uses a shaking mixture of carbon bisulphide, flowers of sulphur and powdered neutral soap (7:1:2). The carbon bisulphide makes the skin tender, and its use necessitates great caution on account of the danger from fire. Uebele found the effect of this treatment uncertain. Instead of carbon bisulphide less poisonous and non-inflammable vitran (with Peruvian balsam āā) may be used, especially if the disease is limited.

Of other methods of treatment the following deserve to be mentioned: The method of Brusasco in which the animal is bathed in liver of sulphur solution (200 gm. of potassium sulphuratum in 70 liters of water); on the following day a third of the body is dressed energetically with a diluted ointment of cantharides (1:6 parts of lard) and on the sixth day the animal is bathed again in liver of sulphur solution. After an interval of 3 to 4 days the treatment is repeated until cure results. Méginn also recommends sulphureted potash baths daily during the first month; during the second month every 2 or 3 days for the space of a quarter of an hour; the same author proposed also a mixture of 100 gm. of flowers of sulphur, 200 gm. of unslaked lime and one liter of water, to be rubbed into the affected skin with a sponge. Lesbrie bathes the diseased animals in liver of sulphur solution, then washes it several times a day with lukewarm cresol emulsion, finally dressing the diseased places with the following ointment: 20 gm. of naphthol, 0.25 gm. of corrosive sublimate and 100 gm. of lanoline. Cadéac paints the previously scarified skin with tincture of iodine, while Dupas treats acariasis with 96% alcohol in a similar way as Schindelka with carbon bisulphide. On the contrary Altmann often saw definite cure result from repeated applications of petroleum (in extensive disease only half the body may be treated at one time). Nicolas treats the affected animals with injections of a 2 to 2½% carbolic acid solution into the depths of the skin (not under the skin), both in the region of the affection and also in the neighborhood; the good effect of this treat-



ment in local acariasis was noted several times by the authors. Moussu found Tessier's mange bath (see page 953) effective in cases of acariasis which were not very extensive; the animals are rubbed for 2 or 3 minutes with brushes while in the bath and kept from licking their bodies; cure resulted after a month. Roth recommends operative treatment in cases of circumscribed disease, which consists in cutting the epidermal layer horizontally with a sharp knife until drops of blood appear, the animal being narcotized; the places are then disinfected, and powdered with xeroform; a bandage is applied and renewed after two days; healing occurs after 5 or 6 days. Eber has also seen favorable results from this procedure. In septicemic symptoms Uebele saw surprising results from collargol clysters (0.5:50 parts of lukewarm physiological salt solution).

In all cases the treatment is to be continued long enough until no fresh nodules or pustules arise and the skin regains its healthy appearance. During the whole duration of the treatment the animal must be nourished intensively, the stable must be kept clean, and care must be taken that the treated parts of the body are not licked. The affected animals must be separated from the healthy ones, and after their recovery kept under observation for a month; on the appearance of suspicious signs immediate treatment must be undertaken. Acariasis of the other animal species may be treated in the manner recommended for sarcoptic scab. Schenzle was successful in a horse affected with general squamous acariasis with caraway oil solution (according to Gmeiner) while in Walther's cases a protracted treatment with a mixture of tar, glycerine and alcohol was necessary.

Considering the all too frequent failure of the treatment, Fröhner and Schindelka consider that acariasis of the dog should be included in those diseases which are under legal supervision.

**Literature.** Altmann, B. t. W., 1907, 41.—Brandl & Gmeiner, W. f. Tk., 1900, 37.—Bügge, B. t. W., 1900, 522.—Cadéac, J. vét., 1906, 80.—Csokor, O. V., 1879, LI, 132.—Dupas, Bull., 1906, 457.—Fettick, Z. f. Tm., 1901, V, 291.—Galtier, Rev. gén., 1907, IX, 22.—Gmeiner, Z. f. Tm., 1909, XIII (complete Lit.).—Grimm, S. B., 1884, 103.—Guinard, J. vét., 1890, 586.—Horneck, B. t. W., 1901, 60.—Legrain & Régulato, Arch. de Parasitologie, 1903, VII, 3.—Mégnin, Les parasites et les maladies parasitaires, 1880.—Nicolas, J. vét., 1907, 538.—Öhl, B. t. W., 1892, 602 (Lit.).—Pfeiffer, B. t. W., 1903, 155.—Prietsch, S. B., 1885, 89; 1903, 81; 1908, 77.—Rieck, *ibid.*, 1900, 53.—Rónai, Vágóhídi Szemle, 1905, 8.—Roth, B. t. W., 1899, 38.—Schenzle, *ibid.*, 1909, 791.—Walther, *ibid.*, 1908, 691.

### 23. Other Mites in the Domestic Animals.

1. **Ticks.** (Ixodida.) Besides mange and hair sac mites, the ticks are the most important temporary skin parasites in the mammalia. They live in the open in nature, and the females especially, if gaining access to the skin of animals, bore into it with the rostrum.

The most important varieties are:

*Ixodes ricinus* (dog tick) which attacks hunting dogs from the month of May to October, but is only exceptionally found in sheep and cattle.

*Ixodes reduvius*, parasitic on sheep and cattle, now and then also on dogs, in the latter animals the tick, the larva or the nymphæ bore firmly into the skin.

*Ixodes bovis* (*Margaropus annulatus*, *Boophilus bovis*, *Rhipicephalus annulatus*, cattle tick) causes inflammation of the skin and leads to the formation of nodules in the skin.

Besides these a number of ticks occur at times in the domestic animals, which like the latter have a practical importance, especially in the transmission of piroplasmosis, and, according to their development are divided into four groups (see Vol. I).

The removal of ticks from the bodies of domestic animals is best brought about by moistening with salt water, oil of turpentine, benzine, petroleum, carbolic oil or simply with fatty oils; but they may be removed by cautious regular extraction without these remedies. By forcible traction the mouth portion may be left behind and cause suppuration or extensive dermatitis, especially in horses and dogs.

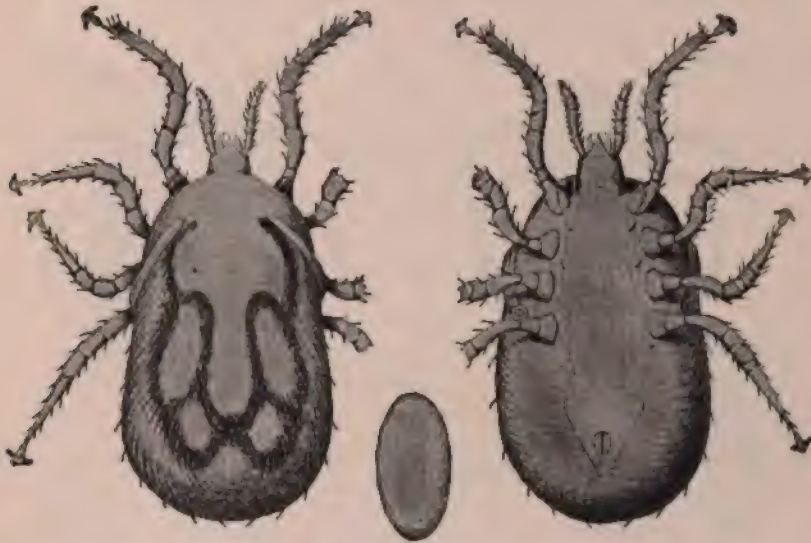


Fig. 157. *Dermanyssus avium*. Female, on the left from the back, on the right from the abdominal side; in the middle an egg. Magnif. 75. (After Mègnin.)

2. *Laminosioptes cysticola*. (*Sarcoptes cysticola*.) It is a minute parasite with longish body, eight legs, and two long bristles on the hind end of the body. The parasites live on the skin of fowls, turkeys, pheasants, etc., and chiefly in the subcutaneous cellular tissue of old animals, where they develop millet-sized nodules which later on become chalky. If they are limited to the surface of the skin only, desquamation is produced. They cause the animal to fall away in condition if they are present in very large numbers.

3. *Dermanyssus avium*. (Fowl mite.) It is yellowish white, blood red if gorged with blood, readily visible to the naked eye, with four pairs of legs at the thorax (Fig. 157). It lodges by day in the crevices of fowl houses or stables, in cages and the



nests of the hens or other birds, while at night it wanders onto the body of the bird (fowls, turkeys, pigeons, geese, cage birds, pheasants) where it sucks the blood, and disturbs the peace of the animals. Sometimes, however, the mites also gain access to the bodies of the birds in daytime, especially into the ear passage, and exceptionally one finds them in great number all over the body. They are very tenacious of life and may remain alive for several months without nourishment; according to Mégnin, their eggs will resist a temperature of  $120^{\circ}$  C.

Fowl mites cause anemia, cachexia, loss of feathers by the unrest and abstraction of blood which they produce, and prejudice the production of eggs in the hens. Not infrequently birds die completely exhausted. Now and then the mites settle also in the nostrils and in the external ear passages, when the birds run about as if wild and swing their heads about (Klee).



Fig. 158. *Leptus autumnalis*. On the left, from the abdominal side; on the right, (gorged) from the back. (After Mégnin.)

In such cases the animals die after a short illness. Otto saw mites occur in immense numbers in young hens where not only the whole body but also the larynx and trachea were affected.

From fowls they may also pass to mammalia (cattle, horses, dogs, cats) especially in stables where chickens are kept. They cause very severe itching as well as skin rashes. Even man may now and then be attacked by the mites. They may also penetrate the external ear passage of cattle, and if they are there in great number they cause restlessness and attacks of dizziness. According to Trouessart and Freund the last mentioned cases are *Railletia auris*, which was designated as *Gamasus auris* by Leydy and others.

**Literature.** Freund, Zool. Jahrb., 1910, XXIX, 313 (Lit.).—Goodall, Vet. Rec., 1906, 251.—Klee, D. t. W., 1901, 3.—Möbius, S. B., 1880, 78.—Otto, S. B., 1908, 45.—Trouessart, C. R., 1902, 806.

4. *Leptus autumnalis*. (Autumn grass mite.) It is the larvæ of *Trombidium holosericeum* (Fig. 158) and lives free in



the fields and shrubs; its oval mite-like body is furnished with three pairs of legs. It occurs in fowls in the summer and autumn, in the region of the roots of the feathers. Although the parasite only remains on the body of the animal for a few days, it causes the formation of little red pustules, and also itching, which is so intense that fowls, especially young birds, may have epileptiform spasms and die in a few days. More rarely the parasites cause little red nodules and pustules upon which reddish colored mites are found; the eruption may occur especially on the head, the inner surfaces of the thighs, the lower chest and abdomen, also in the region of the genital organs, and at times over the whole body (Roth, Liebert); later on bald spots develop, accompanied usually by moderate itching. (The assertion of Roth that dogs are frequently infested with *Leptus autumnalis*, is only true of certain localities.)

In horses the parasite at times causes an inflammatory eczematous condition.

**Literature.** Liebert, D. t. W., 1909, 501.—Roth, W. f. Tk., 1906, 341 (Lit.).

5. **Argas reflexus.** (*A. marginatus*, mussel-shaped seam tick.) An ovoid parasite 6 mm. long, blackish colored in the middle, at the edges yellowish (Fig. 159), which is prevalent all over Europe and extremely tenacious of life, being able to live and even to multiply from 14 to 24 months without sucking blood in the interval (Railliet, Ghiliani). It lives in the day in the crevices of pigeon cotes, but at night wanders onto the body of the pigeon and sucks blood. It attacks young birds especially, which may die after 8 to 15 days in consequence of unrest and loss of blood. Its larvæ also remain on the body of the birds for a long time, or do not leave them at all. At times chickens, ducks and geese are attacked. It may even infest the body of persons attending pigeons and causes painful bite wounds as well as edematous swelling on the skin. The parasite transmits the spirochaetosis of fowls in Bulgaria and on the Island of Cyprus.



Fig. 159. *Argas reflexus*. Slightly magnified. (After Azary.)

The **Argas persicus** occurs in Persia and also in Tunis, and is the intermediary of the spirochaetosis of chickens. The birds are affected by this mite in the same manner as by the *A. reflexus*. According to Carré, its larvæ live very frequently on chickens in Persia. After a slight invasion the parasite is found almost exclusively on the inside of the thighs and wings, whence it passes on to the sides of the body. After an intensive invasion one finds it all over, except in the region of the eye, and then they form mulberry-shaped groups, giving the appearance as if



shot were imbedded under the skin. If no treatment is undertaken the birds die in large numbers.

**Literature.** Carré, Bull., 1909, 172.—Galli-Valerio, Cbl. f. Bak., 1909, L. (Orig.). 189.

**6. Varieties of Trombidium.** Of these the following occur in birds:

*Harpirhynchus nidulans*, which lives in the feather bulbs or in nodules and in little cysts of the skin, and may generally be found there in large numbers.

*Syringophilus bipectinatus*, which is parasitic on chickens and pigeons, and *Syringophilus uncinatus* which lives on peacocks; it lodges within the quills of the feathers; the feathers become lusterless and stunted, the quills lose their transparency and contain in the center a yellowish gray dust-like mass, in which very many mites are visible under the microscope; the affected feathers finally fall out.

**Analges varieties** or feather mites are similar to mange mites, and live in the plumage of the feathers as well as the quills, exceptionally also in the subcutaneous connective tissue; they are either quite harmless or cause hardening of the feathers, and at times emaciation of the birds. They cause changes in the feathers similar to those brought about by the *syringophilus* varieties.

**Treatment.** For the purpose of removing parasites from the bodies of the birds sufficient sand or a mixture of sand and ash must be provided in order that the birds can bathe in it at will (to the sand ash mixture 2 to 5% of flowers of sulphur may be added or finely powdered aniseed or parsley seed). Besides, the bodies of the birds may be sprinkled freely with remedies which have an intense odor and an anti-parasitic action. Peruvian balsam and styrax (in 20 to 30 parts of alcohol), also fennel, aniseed or rosemary oil (diluted with 20 to 50 parts of water or oil, especially for small birds) are appropriate for this purpose. Finally insufflation of flowers of sulphur or pyrethrum powder between the feathers may be recommended, the bases of the feathers being previously smeared with a little soap in order that the powder may adhere better and the insect powder act more energetically. The autumn grass mite has been removed by Liebert by means of bathing the animals once with 1% lysol solution. According to Carré the larvæ of *Argas persicus* are easily removed by painting with oil of turpentine or kerosene.

Since the enumerated ectoparasites mostly exist outside of the animal body in chicken houses, cages, pigeon cotes, etc., and many only attack the birds at night, a thorough cleaning and disinfection of the abodes and removal of coops and birds' nests from the cow barns and horse stables are necessary for the destruction of the mites. The walls are scraped, the wooden parts planed and washed with hot water or hot sodium lye, or are scorched. After all crevices in the walls have been stopped up or done away with, the walls are lime-washed with milk of lime, calcium chloride, or a mixture of lime and creolin, and lime is sprinkled on the floor of the barn. The development of chlorine or formalin vapors may also be considered. Bird cages are best scalded, but in case of necessity they must be burnt. It is also well to wrap the ends of the perches with tarred tow; paint hollow roosts with anti-parasitic fluid; supply the roosts in

cages with reeds into which the mites creep in the daytime, when they can be destroyed.

## 24. Insects Parasitic on the Domestic Animals.

Of the class of insects belonging to the Hexapoda, several species of the orders of Rhynchota, Hemiptera, Diptera, and Aphaniptera as well as Hymenoptera are known to cause more or less severe disease. Their injurious influence is exerted in various ways. By creeping around on the surface of the skin they irritate and hinder the animals from eating, especially on pasture, and in weakly individuals this may now and then have serious consequences. Some insects nourish themselves on the sweat or blood of their hosts, others inoculate acrid substances into the skin or into the mucous membranes of the natural openings of the body, causing acute edematous swellings, which sometimes materially interfere with eating and breathing, but at times produce severe general symptoms of poisoning. At the bitten or stung spot deep seated changes may occur in the skin or subcutaneous connective tissue, such as pustules, hemorrhages, abscesses, etc., which, on account of the itching usually present, lead to traumatic artificial eczemas with the well known results. The larvæ of some varieties establish themselves on raw surfaces, in the skin, and in the subcutaneous connective tissue, or in the body cavities, occasion persistent and even severe tissue changes, and in this way may cause the death of the animals. Finally, certain varieties may bring about the spread of infectious diseases by inoculating bacteria or protozoa adhering to their bodies into the skin wounds.

In the following pages these parasites will be discussed briefly, while detailed descriptions will be left to zoological works.

### A. Hemiptera s. Rhynchota.

(a) **Lice.** (Pediculida.) Parasites generally occurring in badly nourished or cachectic animals, the females of which glue their pear-shaped eggs (nits) to the hairs. They suck blood, cause itching and, when present in great number, lead to a further decline in condition. They may multiply immensely, especially in young cattle during the long stabling in winter, from neglect of skin hygiene, and now and then cause extensive eczema. In Germany lice occur more frequently in military horses, especially in the eastern provinces where much opportunity for contagion from civilian horses is present (Wöhler).

In the domestic animals several varieties of blood sucking lice (*Hæmatopinus*) occur; thus in the horse *Hæmatopinus* s. *Pediculus* *equi* s. *macrocephalus* (Fig. 160), in cattle the *H. eurysternus* and the *H. tenuirostris*, in calves the *H. vituli*, in goats the *H. stenopsis*, in dogs the *H. piliferus*, in pigs the *H. suis* s. *urius*. The *Hæmatopinus suis* is the largest existing louse, its length reaching 4.5 mm.; very



young pigs in which lice are numerous on the skin may die as a result of unrest and disturbance of sucking (Sequens saw 40 head die out of 140; sarcoptic scab might, however, have also been present at the same time).

(b) **Hair Insects and Feather Insects.** (Mallophaga.) These are parasites similar to the preceding ones; they do not suck blood, but eat epidermal scales with their pincer-shaped jaws and gnaw through the fine hair. The females also glue their eggs to the hairs. They are mostly parasites on the head, neck and legs of animals, and cause itching and perhaps also eczematous inflammation of the skin.



Fig. 160. *Haematopinus equi*.  
Magnif. about 25. (After Kitt.)

The varieties occurring in the domestic animals are: *Trichodectes pilosus* in horses, *Tr. scalaris* in cattle (Fig. 161), *Tr. climax* in goats, *Tr. sphaerocephalus* in sheep (sometimes when present in great numbers they produce itching, causing the animals to rub their bodies vigorously and gnaw them; the wool falls out and the affection may then be mistaken for scab); *Tr. latus* in dogs, lastly *Tr. subrostratus* in cats.

The number of feather insects occurring in domestic birds is very large. They disturb the rest of the animal and cause loss of condition when present in large

numbers. They comprise (according to Zürn):

In chickens: *Goniocotes hologaster*, *G. dissimilis*, *Lipeurus variabilis*, *L. heterographus*, *Menopon pallidum*.

In ducks: *Docophorus ieterodes*.

In geese: *Docophorus adustus*, *Lipeurus jejunos*, *Trinotus conspurcatus*, *Tr. squalidum*.

In pigeons: *Goniocotes compar*, *Lipeurus bacillus*, *Colpocephalum turbinatum*.

In turkeys: *Goniodes stylifer*, *Lipeurus polytrapezius*, *Menopon stramineum*.

In peacocks: *Goniocotes rectangulatus*, *Goniodes falciformis*, *Menopon phaenostomum*.

(c) **Bedbug.** (*Acantha lectularia*.) These pass at times on to birds, fowls and pigeons which are kept in rooms or near to dwelling houses (Lucet).

**Treatment.** Lice and hair parasites are killed by various anti-parasitic remedies and removed from the body of the animal. According to the experimental investigations of Bühler creolin liniment (creolin and soft soap, of each 1 part, alcohol 7 parts) ranks first of all on account of its astounding effect as well as its cheapness and slight toxicity; the liniment is rubbed into one-third of the body at a time, and after the third day a bath in  $\frac{1}{2}$  to 1% liver of sulphur solution is given. In severe invasion the process must be repeated 2 or 3 times. Cresol or tar liniment is also very effective, but less innocuous, as is also carbolic acid solution (5%); the somewhat more effective Peruvian balsam is comparatively expensive. Washing with 2 to

3% creolin, lysol, cresol or bacillol solutions has a good effect in every variety of animal but must be repeated several times. Gross recommends for horses a 6%, others a 10 to 15% creolin emulsion, while Schindelka always had good results with a 0.2% creolin solution, which made the employment of other measures superfluous. Two per cent solutions probably answer best, but must be employed repeatedly. Gray mercury ointment, which is not particularly reliable, may also be employed in mild cases; a piece about the size of a hazelnut may, in the case of horses, be placed on the inner surface of the harness, in cattle between the horns, in dogs half the quantity on the inner surface of the collar, but in all animals it is most properly rubbed on the diseased parts of the skin. On account of the danger of mercury poisoning the employment of mercurial ointment must be avoided if several animals in a stable, particularly young ones, must be treated. Widespread louse infection is not removed by partial applications of mercury ointment. Fröhner obtained very rapid results by cautious washing with a 1% corrosive sublimate solution. With tobacco decoction (1:20-25) which is also effective, the body should be washed only in sections where there is general disease in horses and cattle, in order not to run any danger from nicotine poisoning. In dairy barns where preparations containing tar or cresol cannot be used, because their smell is taken up by the milk, Schindelka recommends washing with decoction of wormwood or of *chrysanthemum inodorum*; decoction of *stavesacre* (sem. *staphisagriae* 5:100) may also be employed, but according to Bühler the effect of this drug is not very reliable. The same is true for the Persian insect powder (*flores pyrethri*) and other insect powders which exert a parasiticide effect only after previous moistening of the animals or if applied in form of a paste; they are applicable chiefly in small sucking animals and birds. Petroleum with linseed or rape oil also kills the parasites, but in fine-skinned animals it sometimes causes dermatitis. Good effects are also produced by ethereal oils (1:10 alcohol or water), and by benzene water. Fatty oils or fats have been used successfully.



Fig. 161. *Trichodectes scalaris* (on a hair). Magnif. about 25. (After Kitt.)

The so-called vinegar of arsenic (arsenic and potash of each 15 parts, vinegar and water of each 1500 parts) suggested by Viborg and Schleg exerts a prompt effect but is dangerous, especially in extensive disease, and therefore is to be avoided.



Two or three repetitions of the treatment after 3 to 4 days appear to be indicated in order to destroy the parasites as they hatch from the eggs. By washing with vinegar the eggshells will be dissolved and the embryos killed. Clipping the hair materially facilitates the destruction of the parasites; in horses, however, it should (according to Wöhler) be undertaken only if numerous parasites are present, since the soaked hair prolongs the effect of the anti-parasitic remedy. In winter the treatment of a large herd of horses may have to be omitted, the measures being directed only to the prevention of a further spread of the parasites by treating and isolating the manifestly affected horses.

Against feather mites the parasitocides may be employed, which have previously been recommended as suitable for birds (see page 982). Careful cleanliness and skin hygiene afford the most effective protectives against lice and similar parasites in general, and play an important part in the treatment. If the affection obtains a considerable extension, disinfection of the barns and of any objects with which the affected animals come in contact is indicated.

**Literature.** Bühler, Experm. und klin. Unters. über Wert und Wirkung d. Kreolinlinimentes, Diss. Giessen, 1909 (Lit.).—Wöhler, Z. f. Vk., 1906, 219.

## B. Diptera. (Flies.)

(a) **Long antennaed** (Nematocera). These are small insects whose larvæ develop in water, damp earth or shady places; in the mature condition they feed on plant juices, but the females of some varieties also attack domestic animals and suck blood.

Of the **gnats** the following are noteworthy:

The stinging gnat (*Culex pipiens*) prefers the skin of man, but now and then also stings animals and produces painful swellings on them.

The sand flies (*Simuliæ*) are much more important in this respect. The *Simulia reptans* and the *Simulia cinerea* cause inflammation on the thin hairless parts of the skin of the horse, for instance on the inner surface of the thigh, on the internal upper surface of the ear, which only heals gradually and with desquamation. The *Simulia reptans* appears at times in great swarms and causes fatal results. Thus in May of 1881, in 10 townships of an Upper Hungarian county, 37 head of cattle and 4 horses died as a result of their bites. In Sweden (Schonen province) gnats occur especially in hot summers and attack cattle, horses, and also sheep. The *Simulia ornata* may also occur in similar swarms (in one instance out of 170 cattle that were stung, 27 died), and, according to Dammann & Oppermann, may transmit the infection of hemorrhagic septicemia in deer and cattle. According to Wigand one observes severe circulatory disturbances and falling of temperature in animals poisoned by



gnat stings; also edematous swellings always on the abdominal portions of the body where the skin shows round spots similar to flea stings.

The treatment and prophylaxis are similar to those used against the *Columbae's* fly.

*Columbae's* fly (*Simulia maculata* s. *Columbaesensis*), 3 to 4 mm. long, posterior ash-gray, on the sides and underneath colored yellow, 3 black stripes on the back, with black spots on the abdomen.

The female lays her eggs at the end of May and beginning of June in the wooded regions of the lower Danube in the water of the mountain brooks; they take the form of slimy masses, from which the larvæ are hatched in 2 to 3 weeks. With a clinging apparatus situated at the hind end of the body these adhere to stones and plants in the water, until after several moultings pupæ emerge which remain torpid during the winter, but the following year, towards the end of the month of April they emerge as fully developed gnats (*Tömösváry*).

The *Columbae's* fly owes its name to the fact that in certain years it increases enormously in the neighborhood of the Servian fortress of *Columbae* on the lower Danube, and from here spreads in great swarms, and for long distances. As a rule several swarms follow each other, the first about the middle of April, the second the beginning of May, the third about the middle of May; the last swarm which consists of the last hatching, occasions only little injury. They occur in great numbers mostly in the warm spring after a mild winter, while torrential rain, great droughts and severe spring frosts considerably interfere with their development. The swarms collect before sunrise and follow the direction of the wind, leaving the Danube pass above *Báziás* and are then driven by the wind in different directions (*Tömösváry*). They appear at times in immense numbers in southern Hungary, especially in the counties of *Torontál*, *Temes* and *Krassó-Szörény*, also *Arad*, *Csanad* and *Hunyad*, in *Servia*, the swarms form dark clouds and may cause a very large number of fatalities especially among cattle, but also among horses and other domestic animals. In such years the swarms sometimes go further north to *Austria* and *Germany*, but seldom cause death in these countries.

In the County of *Temes*, 52 horses, 131 cattle, 316 sheep and about 100 swine died in the year 1783, while in the year 1830 several hundred horses and cattle perished. In the year 1813, 200 head of cattle in *Arad*, and in *Versecz* 500 head were killed by the gnats. In the year 1880, 400 pigs, 80 horses and 40 cattle died in *Kubin* within 4 hours, and in the county of *Hunyad* about 100 cattle, 5 horses and 80 pigs. Great swarms were last noticed in the years 1888, 1889, 1895. In the year 1895 in the county of *Hunyad* alone from May 21 to May 31, 6 horses, 274 head of cattle, 53 sheep, 66 goats and 92 pigs died and the loss amongst wild stock was also considerable. In the southern parts of the county the gnats cause more or less considerable losses every year.

On the approach of the swarms the animals become restless and seek refuge in the stables or in the water. The attack on animals that are kept in the open occurs by immense numbers of gnats, the females preferably injuring the regions of the mouth, nose, eyes, genital organs and rectum with unnumbered stings; through the natural openings they also penetrate into the interior of the body and attack especially the mucous membrane of the throat and larynx. At the seat of each sting a small painful swelling develops, from the coalescence of which extensive tumefaction occurs; those around the nasal openings and pharynx interfere with the breathing, and as a result death may occur in 5 to 24 hours. In the production of the fatal result there is no doubt that the poison inoculated by the flies plays a part. In a few cases the swellings disappear after 2 to 3 weeks; for some days the animals eat badly or not at all, and



therefore they become weak and their temperature rises. Kemeny noticed blindness and a bounding heart beat in 2 horses; the blindness persisted later on. (The gnats also attack the skin of man, especially that of women and children, and infants may die as a result of the stings.)

The postmortem examination of dead animals shows an edematous infiltration of the subcutaneous or submucous connective tissue at the spots corresponding to the swellings, and the sting punctures on the skin or mucous membrane may be recognized as bright red points. Now and then one finds dead gnats in nose, larynx and trachea. The autopsy findings indicate death by suffocation; the spleen is often acutely swollen.

Apart from housing the animals in dark places, the treatment consists in cold compresses or general spongings, also in rub-downs with ammonia, acetate of lead or alcohol; besides carbolized or salicylated oil (10%) or a mixture of lime water and oil may render good service. In case of heart weakness cardiac remedies may be used. Wigand administers  $\frac{1}{2}$  to  $1\frac{1}{2}$  liters of brandy at one dose, or  $\frac{1}{4}$  to  $\frac{1}{2}$  a liter at intervals of 1 to 2 hours until improvement has occurred.

Since a radical extermination of the gnats or even a limitation of their numbers appears impossible, the safest prophylactic measure consists in housing the animals in dark stables at the time of the appearance of the swarms (from April 20 to 10-15 of June) and only letting them out to pasture at night. If it is necessary to give the animals their freedom during the day then it is advisable to produce thick clouds of smoke by burning manure or damp weeds, and thus to ward off the gnats from the herd (the production of smoke is also advisable in front of the stable door). Besides, working animals may be smeared on the thin-skinned parts of the body, e. g., around the natural openings, with any stinking mixture, such as inspissated tobacco decoction to which fat and petrolenm may be added (Schönbauer), also with assafetida, tar, naphthalin or iodoform, etc.

(b) **Short Antennaed Flies** (Brachycera). Mostly large insects, with three short antennæ consisting of three joints. The following families are parasitic on the domestic animals and suck the blood:

a. **Gadflies** (Tabanida); large flies living in woods and on pastures, which attack large animals on hot summer days, penetrate their skin, suck their blood and thus harass them; after they have left the animal a drop of blood oozes to the surface of the skin from the punctured spot.

Among the numerous varieties the cattle gadfly (*Tabanus bovinus*) a fly 27 mm. long is most frequent, and principally annoys cattle; similar flies are the *T. autumnalis*, *T. morio*, *T. tropicus*, the *T. lineola* and others.

**Haematopota**; narrow, longish flies with white-dotted wings; their chief representative is the rain gadfly (*Haematopota pluvialis*) which harasses the animals on the approach of stormy weather.

The blinding gadfly (*Chrysops coecutiens*) is a fly 9 mm. long with broad spreading wings and sharply defined abdomen; it flies especially about the head of the animals, and occasionally causes inflammation of the eyes and ears.

**b. Flies (Muscida)** in the restricted sense. Most insects belonging to this class merely annoy the animals and hinder them from eating at pasture or they cause waste of food. Some of their representatives also cause considerable disturbance of health.

In inhabited places the house fly (*Musca domestica*) is frequent everywhere; it occurs in large numbers in stables and in the brushwood about meadows; other flies are the *M. corvina* which sucks the sweat, the *M. vomitoria* (vomiting fly), the *M. cadaverina* which is parasitic on dead animal bodies, the *Sarcophaga carnaria* (meat fly) and the *Sarcophaga magnifica*, the female of which deposits its larvæ, that are born alive, on wounds and swellings as well as in the ears, and in the vagina of female animals. In a case observed in a goat by Wirth the larvæ of the *Sarcophaga magnifica* were found in a necrotic patch of the vagina; fever was also present.

The larvæ of *Lucilia cæsar* and *L. sericata*, which belong to this class, produce the fly larvæ disease of lambs; this is prevalent especially in Holland and New Zealand. The larvæ of these species of flies are hatched from the eggs that have been deposited in the perineal region of lambs suffering from diarrhea. They cause violent itching, wander from here to the root of the tail and to the sacral region, where they riddle the skin and produce an inflammation which still more exhausts the weakened animals. According to Gilruth the larvæ at times pass through the abdominal wall into the abdominal cavity. The fly larva disease was observed by Gaber in Germany (Pr. Mt., 1877, 78, 82) and by Jordal in Norway (Norsk Vet.—Tidskr., 1905, 121).

In America the larvæ of *Lucilia macellaria* establish themselves on wounds, whose healing process they disturb, and even wander into the nasal cavities. According to Jerwolajew (B. t. W., 1905, 676, Rev.) the "Welfare fly" in Russia deposits its eggs on wounds on sheep and cattle, where the liberated larvæ destroy the tissues, and at times this destruction extends so deeply in the umbilical region as to produce hernia.

The treatment consists in removing the larvæ of the flies with forceps, applications of 5% carbolic acid or creolin solution, in painting with 5% carbolated oil, or in the introduction of creolin suppositories (creolin 1 part and cacao butter 20 parts). The removal of flies from the stables is attempted by establishing conditions unfavorable to their life (sharp draughts, partial exclusion of daylight, fly nets, etc.), by mechanical destruction of flies and their broods and by keeping off the flies from animals in the open (by blankets, etc., by substances having an objectionable smell).

The stinging fly (*Stomoxys calcitrans*), belonging to the same family, takes up its abode in stables and may be recognized because it rests on the walls with the head directed downwards; it attacks the feet of horses,



causing the animals to kick and stamp. The *Haematobia* species are much smaller and found especially out in the pastures (*Haematobia stimulans*, *H. ferox*, etc.); they disturb animals when grazing.

The tempest fly (*Hydrophobia* s. *Anthomyia*) is a small fly which, on the approach of rain, flies in great numbers around the eyes and noses of cattle. The tsetse fly (*Glossina morsitans*) resides in the swampy regions of Africa and transmit the trypanosomes of Nagana by its bite (Vol. I).

c. **Bot Flies** (*Estrida*). These large flies pass through one period of their development in the bodies of animals as larvæ, consisting of 12 segments, and here cause more or less severe disease, according as they localize in the cavities of the facial bones, in the stomach, intestines or in the subcutaneous connective tissue. The completely developed larvæ leave the body of the host and change in the earth to barrel-shaped pupæ. The female of the gadfly developing from these pupæ harasses the larger animals on warm summer days.

The *Æstrus* (*Hypoderma bovis*) is a black fly, 13 to 14 mm. long, with gray head and brownish narrow wings which stand out from the body. It swarms at the beginning of summer and into the autumn, more especially from June to September, but produces no harassing buzzing and does not disturb the animal, nor does it cause them to be stampeded, as many assume. This stampede is much rather caused by other insects, principally by *Tabanidæ* (Ostertag, Ströse, Hoffmann).

The development of the *æstrus* is not yet known in all its details, although many investigators have occupied themselves with its study (Brauer, Hinrichsen, Ruser, Koorevaar, Hoffmann, Ostertag, Jost, Ströse). The pairing of the flies takes place on high mountain tops, sunny rocks, towers and other high lying places. Nothing definite is known of the manner in which the eggs are deposited. Since hitherto no eggs of the *æstrus* have been found on the animal host, the first assumption would be that the female deposits her eggs on grass, and that later the eggs themselves or their larvæ are swallowed by the cattle when grazing (Hinrichsen). But on the other hand it may easily be possible that the *æstrus* deposits its eggs on the bodies of cattle, where the eggs cannot be found, being hidden in the depths of hair or because the larvæ, which are transparent and therefore not readily recognized, are hatched in a very short time.

The penetration of the embryos into the bodies of cattle probably takes place in great part or exclusively by the digestive canal in such a manner that either the eggs or the larvæ of the fly are swallowed by the animal, or according to Ostertag, the larvæ reach the cavity of the mouth by active wandering. Ströse on the contrary considers it possible that some of the larvæ penetrate through the skin into the subcutis, their powerful chitinous mouth apparatus rendering them quite capable of doing this.

For the correctness of the last named assumption it may be stated that wandering larvæ only rarely are found on the way between the esophagus and the subcutis, further that the youngest larvæ in the subcutis correspond morphologically to the esophageal larvæ and that larvæ are often found simultaneously under the skin

and in the esophagus in the like stage of development. In favor of this view observation shows further that thin-skinned young cattle more frequently carry fly bites than other cattle, that in man the uncovered parts of the skin mostly form the dwelling places of the larvæ, and finally that Brauer has observed the penetration of the larvæ of *Estromyia satyrus* into the skin of guinea pigs and rabbits. The circumstance that in autumn and at the beginning of winter larvæ have not yet been found under the skin might be because the discovery of the small and transparent larvæ in the subcutaneous cellular tissue presents insuperable difficulties.

The wandering of the larvæ takes place variously according to their mode of penetration into the body. The swallowed larvæ bore through the mucous membrane of the esophagus and pharynx, and are found in great number in the submucous connective tissue of the gullet from the months of July to November. Thence they wander along the blood vessels and nerves in the direction of the spinal column, and even pass through the openings between the vertebrae into the spinal canal, and mostly remain there in the dural fatty tissue, from the month of December to March; from January to July they get into the subcutaneous connective tissue which they may do without first entering the spinal canal. The larvæ, which accidentally bore through the skin a short time after the eggs are deposited, penetrate into the subcutis at the point of their deposition without any wandering about. After two moultings and therefore three stages of development have been gone through, they emerge from the swellings which they have produced, become pupæ in the ground in the course of 12 to 36 hours, and the fully developed insect appears after about 30 days.



Fig. 162. Larva of *Hypoderma bovis*, on the left from the back (concave), on the right from underneath (convex). Natural size.

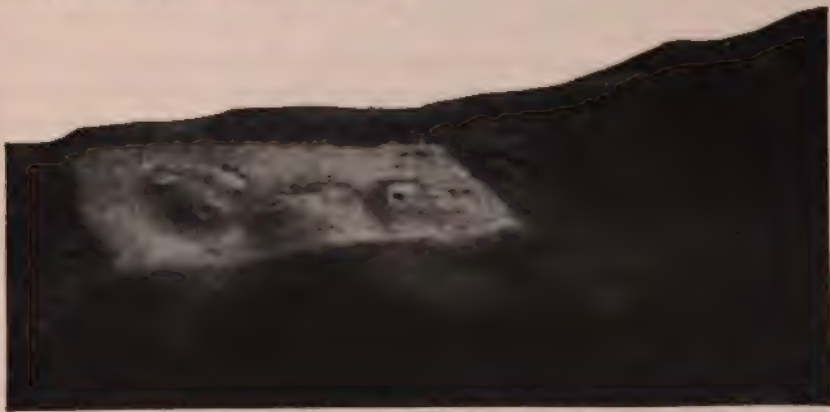


Fig. 163. Bites of *æstrus* in the loin region of an ox.

Experiments of transmission with *æstrus* larvæ have been carried out chiefly by Koorevaar, who placed larvæ obtained from the spinal canal of slaughtered cattle under the skin of a dog and a goat. In 14 days the dog was killed, and all the larvæ introduced were found to be alive, some in the subcutaneous connective tissue, some between the muscles, in the abdominal cavity, in the wall of the pharynx, outside of the trachea, and in the fatty tissue of the vertebral canal. In a goat swellings were visible in the subcutis after 12 days. Similar experiments by Ruser and Jost with esophageal larvæ were negative, but in two experiments by Ströse on a calf the results were positive; the introduced esophageal larvæ became quite mature in 3 to 10 weeks and were found in the subcutaneous tissue.



The occurrence of the œstrus fly is limited to certain localities. Usually one finds the fly from Scandinavia to Southern Europe; further it is prevalent over Asia, Africa, North America and Australia (Ströse). But the local prevalence of the œstrus depends essentially on external circumstances. Thus the fly plague is observed exclusively in those regions where the cattle are out day and night from the beginning of spring on, or are driven there in the morning hours. On the contrary those regions remain free from the pest where no pasturing is done or where the grazing only begins in autumn, or where the cattle are driven out to pasture only in the midday hours (as in many localities of South Germany). Factors that are as yet unknown, perhaps climatic or certain conditions of soil also appear to be of influence, for only in this manner can the observation be explained that certain neighborhoods are avoided by the flies, although imported cattle infested with œstrus swellings remain on the pastures during the whole summer. On the other hand the spread of the fly by cattle with œstrus swellings cannot be denied in all cases (Ströse).

In Germany warbles are most frequently met with in Eastern Prussia, in some districts of Western Prussia and in the province of Posen, in a considerable part of Pomerania, Brandenburg and Altmark, in the greater part of Schleswig-Holstein, in the whole of East Friesland, Oldenburg, in several districts of Westphalia and of the Rhine country.

The œstrus larvæ living in the subcutaneous connective tissue produce nodules which may become as large as walnuts and in which they lie imbedded in pus. In the skin the swelling gradually increases in size and shows an opening which is at first about the size of a pin's head, but later on 4 to 7 mm. broad and circular. This is produced by the boring movements of the larva, which in its second stage of evolution is already provided with a breathing apparatus, and on that account requires air. From the opening at first a sero-purulent secretion issues, which mats the hair in the neighborhood. At a later stage the black-looking hind end of the larva is close beneath the opening and the larva itself may be expressed from the swelling. It is 22 to 27 mm. long, up to 15 mm. thick and according to its development white, gray, or grayish black. The formation of the swellings begins at times as early as in the month of January, generally, however, in the months of February and March, and exceptionally later. They disappear a few weeks after the emerging of the larvæ, from the end of April to the beginning of June, and leave no trace behind them. Isolated warbles can, however, be noticed in the month of July in any neighborhood where pasturing begins late. Now and then the larvæ die under the skin, or after the exit of the larva the cavity of the nodule is filled with granulation tissue in which lime salts may be deposited, and in this way hard nodes persist in the skin (Casparini).



The exit of the larvæ mostly occurs in the early morning hours but also at night, and more rarely at midday and in the afternoon. The assertion of some authors that the exit of the larvæ occurs exclusively in the early hours of the morning is, according to Ströse, not true in all cases. This peculiar behavior of the larvæ is apparently due to the fact that strong pressure is exerted on the warbles on lying down and getting up, and subsequent stretching of the muscular tissue on the back of the cattle (Ströse).

The larvæ of other *œstrus* flies cause lumps similar to those of the *Hypoderma bovis*. Thus in cattle, especially in South Russia, on the Balkan peninsula, in Italy and in Norway, are found the larvæ of *Hypoderma lineata*. The warble fly of Australia also presumably belongs here. The larvæ of *H. actæon* live under the skin in red deer, those of *Hypoderma diana* in the roebuck. On the other hand one finds the larvæ of *Hypoderma bovis* also in warbles of horses and asses. Rips saw warbles, usually along the back, in about 20% of the animals of a large transport of horses; they were found only on damp parts at certain places of the skin. The animals often stretched themselves, they had a cramped gait and edematous swelling of the anterior limbs on account of simultaneous serous inflammation of the connective tissue under the shoulder blade. [Only the *Hypoderma lineata* occurs in the United States.—TRANS.]

In man warbles occur under the skin, especially in Central America, Mexico, and in a large portion of South Africa, only exceptionally in Europe, more particularly in Scandinavia. They belong to the *hypoderma* species which are parasitic in animals, and are chiefly found on uncovered parts of the body.

The importance of warbles depends wholly upon their number. They generally exercise no prejudicial effect on the state of health of the animal, but if present in great numbers (50 to 120) they cause emaciation and a diminution in the ability to work and in the milk supply. Some young cattle even die or become affected with extensive skin edema. A much greater importance attaches to the larvæ in every case through the depreciation of the skin and the flesh, since the skin of the infested animals is either perforated or, after the exit of the larvæ, it contains little cicatrices, and thus its value will be diminished in proportion to the holes or scars, the more so as these changes are mostly met with in the most valuable part of the skin, namely that of the back, loins and croup.

The injury caused through the depreciation of the skin and flesh is calculated in Germany to be at least \$1,500,000 to \$2,000,000 annually, in England up to \$40,000,000 (Ostertag, Ströse), and in Ireland up to \$2,500,000 (Müller). The lessened value of the skin of each head of cattle affected with warbles is put by Ströse as at least \$1 to \$1.50. The diminished value of the flesh of a beef is calculated by Külman at \$7.50 to \$10.00. In Australia also the larvæ seem to occasion considerable injury, and there the disease is notifiable.

With reference to the considerable economic injury the extermination of warble flies deserves full consideration. In neighborhoods where driving the cattle to pasture cannot be

avoided, especially in the morning hours, the warble plague may be combatted by a systematic destruction of the maggots. This prophylactic measure, must, however, evidently be carried out by concerted efforts in affected regions.

The right time for commencing the destruction of the larvæ is from the end of April to the beginning of May, shortly before pasturing begins. At this time the larvæ are not so far developed that they can crawl out from the swellings, but most warble lumps already have an opening through which the larvæ may be removed. The procedure in getting rid of the larvæ is as follows: If the pressing out of the larvæ with the fingers cannot be accomplished, then they must be pulled out with small, narrow forceps, or one punctures the end of the larva lying near the hole with a crochet needle, whereupon the contents of the body of the larva flows out, and the swelling may be evacuated by strong pressure with the fingers. Larvæ which cannot be removed even then may be left in the lumps where they die and gradually suppurate out without usually disturbing the general health of the animal. Exceptionally, however, a phlegmonous inflammation may develop here, or more frequently a nettlerash arises some hours after the evacuation of the warble lump. For the purpose of puncturing and pulling out the larva one may employ a needle supplied with a small barb (after the nature of the crochet needle). The dilatation of the opening of the warble lump with a knife should only be undertaken by a veterinary surgeon. The last named procedure has now and then been followed by very distressing phlegmons (Schmidt). During pasturing, up to about July 1 the animals should be examined about every 14 days for newly arising warble lumps, and any larvæ present must be removed at the same time. The removal of the larvæ is done in the stable or yard if possible.

Closing of the openings of the swellings with heavy oil or tar and the use of ointments only kill a part of the larvæ; the procedure is therefore not suitable for combatting the pests. But the effect of removing the larvæ is supported by suitable care of birds which destroy the larvæ and the warble flies; such birds are the starling, jackdaw, hoopoe, thrush, red-start, wagtail and titmouse (Ströse).

In Denmark some communities engage persons specially for the purpose of removing the larvæ. These examine the herds in their care from 4 to 6 times in spring and early summer, and if warbles are present destroy the larvæ with small steel pincers. In Germany the procedure is also customary in various parts of the country and in the Grand Duchy of Oldenburg the intention is to enforce its practice by law.

The larvæ of other bot flies reside within the body, for instance the larvæ of *Cæstrus ovis* in sheep in the cavities of the facial bones (see page 21), the larvæ of *Gastrophilus equi*, especially in the stomach of the horse (see page 452), the larvæ of *Gastrophilus pecorum*, *G. hæmorrhoidalis* and of *G. nasalis*, also in the stomach or duodenum of the horse (the larvæ of *G. pecorum* also in cattle), (see page 453). The flies annoy the large domestic animals only when laying their eggs.

According to Railliet the larvæ of *Ochromyia anthropophaga* live under the skin of the dog, and cause lentil-sized and painful nodules on the tail, on the ears and the feet.

**Literature.** Jost, *Hypoderma bovis*, Diss. Leipzig, 1907 (Lit.).—Ostertag, Z. f. Flhg., 1906, XVI, 407.—Rips, Z. f. Vk., 1909, 138.—Ströse, Arb. d. G.-A., 1910, XXXIV, 41 (Lit.).—Villemoes, Z. f. Flhg., 1906, XIV, 226.

(c) **Pupipara.** The *Hippobosca equina* occurs frequently, and is a brown fly 8 mm. long, with yellow head, three yellow points on the breast, and longish rounded wings. It annoys horses in summer, and exceptionally also cattle and dogs, by settling in the region of the rectum and vagina, as well as on the thin skin on the inner surface of the thighs.

To this class also belongs the *Melophagus ovinus* (sheep



tick, sheep louse fly), a wingless insect similar to a louse, that lives between the wool, nourishing itself on the sweat, on the wool fibers, and the blood of its host, and molesting the animal by causing itching. The sheep tick harbors in its body the *Critidia melophagea*, a protozoon similar to the trypanosome, which has been discovered in the Pathological Anatomical Institute in Budapest, but its transition into the blood of the animal host has not been observed. To get rid of melophagi the same procedure should be adopted as with lice, namely dipping in tobacco decoction, decoction of walnut leaves in vinegar, solutions of assafetida (2:100) or the use of weak creolin solutions. Friedberger & Fröhner recommended applications of gray mercury ointment (3 to 4 gm.) along the back and below the neck. Shearing also helps materially in getting rid of the parasite. The purification of the stables and subsequent whitewashing with chloride of lime is essential; crevices in the walls especially should be thoroughly cleansed and filled up.

#### C. Fleas. Aphaniptera.

Fleas occur in dogs, cats and rabbits, also in chickens and pigeons, and the flea which is parasitic on man, *Pulex irritans*, may infest the bodies of the domestic animals. The fleas also which reside outside the animal body, especially in the dust and clefts of the ground, settle on young and feeble animals, not rarely in very large numbers and cause intense itching which may lead to the development of severe dermatitis.

Their removal is best effected by diligent care of the skin and by occasional baths. In severe cases the same treatment is indicated as with lice (see page 983).

In America and Africa there lives a far more dangerous flea, viz., *Pulex s. Sarcopsylla penetrans*, which by its bite may cause violent inflammation with subsequent ulceration and necrosis of the skin in men and animals.

#### D. Hymenoptera. (Hautflügler.)

(a) **Bee** (*Apis mellifica*). In the neighborhood of beehives animals are at times attacked by bees when these are swarming, and they may cause death by their stings. At the points of the stings the skin or mucous membrane swells, and dyspnea may occur owing to the swelling of the nasal mucosa and also symptoms of collapse, diarrhea, further hemoglobinemia and jaundice (Albrecht).

**Literature.** Albrecht, W. f. Tk., 1892, 241.—Fünfstück, S. B., 1885, 75.—Ganter, B. Mt., 1905, 10.—Hable, Ö. Vj., 1892, N. F. IV, 96.—Jagnow, Z. f. Vvk., 1898, 22.

(b) **Bumble-bee** (*Bombus terrestris*). This at times attacks horses and cattle if the nests of the insects are rooted up



by the plough (Bissauge). The symptoms are similar to those of bee stings (B. Kovacs saw symptoms similar to those of hydrophobia in a dog [Vet., 1895, 361]).

## 25. Parasitic Thread Worms in the Skin.

The thread worms which are parasitic in the skin and subcutaneous connective tissue of mammalia belong to the family of *Filaria*. Of this class the following varieties are known:

1. ***Filaria hemorrhagica***. A fine, thread-like worm which shows transverse striae on its white body, while its head end is covered with a round papilla-like prominence; the male 28 mm., the female 40 to 70 mm. long. The course of evolution is unknown.

In its mature state the parasite lodges in the subcutaneous and intermuscular connective tissue of horses of oriental descent (Hungarian, Russian, Tartaric) and causes frequent cutaneous hemorrhages in spring and summer, the so-called summer hemorrhage (Drouilly, Trasbot, Mégnin, v. Ratz and others).

The exuding blood lifts the skin or the epidermis in the form of a lentil or hazelnut-sized nodule, which bursts in 1 to 2 hours, whereupon blood exudes for a time from the fine opening thus formed; after a while, however, the bleeding ceases and the slight wound heals. The nodules form as a rule in great number and close together, but only in the warm time of the year. The disease may recur annually in the same horse for 3 or 4 years, but afterwards disappears completely.

Unless severe anemia is caused through the hemorrhage (Brunowie, Liautard), the health of the animals is not affected. The complaint is chiefly annoying, because the exuding blood soils the skin and harness. The treatment consequently consists only in frequent washing and cleaning of the skin, and besides it appears advisable to protect the bleeding region from chafing by the harness wherever possible.

**Literature.** Condamine & Drouilly, Rec., 1878, 1144.—Railliet, Zool. méd., 1895, 507.—Railliet & Moussu, C. R., 1892, 545.—v. Ratz, Vet., 1898, 393.

2. ***Filaria irritans***. The silver white cylindrical larva, 2 to 3 mm. long, of an hitherto unknown thread worm (Rivolta, Laulanié). According to Roger it may be the larva of *Oxyuris equi* (see page 487). It resides in the subcutaneous connective tissue of horses and causes a skin disease described by the name of dermatitis granulosa s. pruriginosa s. verminosa ("Summer sores" [French]). These are noticed exclusively in horses in the summer, and manifest themselves by the formation of little nodules in the skin, where it comes in contact with the harness, and on the legs, also not infrequently on other parts of the body, and even in the conjunctiva of the eyes

(Quéraud); from these nodules ulcers arise, the walls and edges of which are covered with brownish red granulations. Between these, millet-sized to hempseed-sized, yellow, cheesy or calcified foci may be found, each one of which harbors the filarial larva, the white color of which forms a distinct contrast.

The granulating ulcers show a great tendency to further spread and obstinately resist every treatment; this is due in part to the continued rubbing from intense itching. The ulcers generally heal towards the end of the summer, but recur in the following years when the warm weather comes.

A similar complaint (socalled *caloris*), was observed by Schindelka in military horses in Hungary, particularly in the neighborhood of Kecskemet and Debreczen. Gero & Moharos have recently described it under the name of "calore sores." The last named disease, which appears chiefly to attack stud horses, begins with the appearance of circular swellings, chiefly on the body, but now and then also on the limbs, from the surface of which a sticky fluid exudes. This dries, becomes parchment-like and is cast off after 6 to 8 days, whereupon malignant and gradually increasing ulcers form. The process only rarely extends deeply and then may lead to septic infection and death. With the onset of cold weather ulcers which have previously resisted all treatment heal spontaneously. (Two cases of this disease have been observed by Marek, but no parasites could be detected in the ulcers, which already commenced to heal.)

For treatment Rey recommends applications of sulphide of arsenic in a thin layer, whereupon a dry scab, about 1 cm. thick, forms on the surface, which falls off after 8 to 10 days and healing then generally occurs in a short time. Blaise treats the granulating surface with ether, chloroform or iodoform at intervals of 1 to 2 days, and by this procedure produced a cure within a fortnight. Vanuta recommends the use of the red-hot iron, while Darrou saw favorable results from the injection of a 2% sterile solution of potassium permanganate at several places in the region of the wounds (1.0 cc. in 4 or 5 places). Liénaux employed an arsenic paste (acid. arsenic., pulv. sabinæ aa, gummi arab. aqua dest. q. s.). Quéraud found painting with picric acid solution, and also (in the treatment of conjunctival ulcers) copper sulphate very effective. In the so-called *calore sores*, in the cases of the authors, a mixture of 1% picric acid solution and glycerine has answered well.

**Literature.** De Doës, Holl Z. f. Niederl. Indien, 1906, 303.—Gerö & Moharos, A. L., 1906, 493.—Huguier, Bull., 1904, 469.—Liénaux, Ann., 1907, 137.—Quéraud, J. vét. 1907, 621.—Roger, Rev. vét. 1907, 6 (Lit.).—Schindler, Ú. M., 1903, 49.

As *Dermatosis aestivalis buccarum* Ablaire describes a peculiar skin disease of the horse which occurs very frequently in the valley of the Meuse, but is also known in Saumur and Algeria, and also in Africa under the name of "eczéma zébré." After a transitory edematous



swelling has developed, the hair of the submaxillary region becomes bristly and matted by dried droplets of serum. Soon the hair falls out, and narrow whitish stripes arise on the skin running in bow shape, and covered with scales; they itch intensely, and change into "summer sores" as a result of rubbing. Disinfectant fomentations and applications of zinc oxide and camphor ointment proved effective.

While Ablaire assumes nocturnal parasites as a cause of the disease, Langiny believes it to be due to fly stings; Diudonné, Cadiot & Railliet refer it to the presence of *œstrus* larvæ. (The creeping about of the larvæ of *Gastrophilus nasalis* was noticed in the skin of man in Russia.) Nicolas & Cazenave, however, consider the complaint to be a superficial inflammation of the lymphatic vessels in the skin which progresses centrally, yet Petit saw no signs of a lymphangitis in such cases under the microscope. Ablaire, Bull., 1905, 538.—Langiny, Bull., 1908, 279.—Nicolas & Cazenave, *ibid.*, 1908, 287.

3. ***Filaria immitis*.** The embryos of this worm live in the blood of dogs (see Vol. I), while the sexually mature worm occurs in the right half of the heart and in the blood vessels, also in the subcutaneous and intermuscular connective tissue, and either free or enclosed in cysts which one can feel under the skin (v. Ratz found them in great number in the subcutaneous and intermuscular connective tissue of three young Italian greyhounds).

4. ***Filaria bancrofti*.** This thin worm which may attain a length of 50 cm. lives in tropical regions under the skin of man in the lymph vessels, and causes a very great thickening of the legs (elephantiasis Arabum), while its embryos circulate in the blood in large number. Its larvæ live in crustaceæ of fresh water (Cyclops), they are taken up with these in the drinking water, and are set free in the stomach after digestion of the crustaceæ. In the same regions the worm is also encountered under the skin of cattle, horses and dogs, and several worms may frequently be met with at the same time (Piot, Railliet).

Rivolta, later Siedamgrotzky, Schneider and Künnemann each found in a case of circumscribed inflammation of the skin of a dog a lively moving thread-like worm in the contents of pustules examined under the microscope, which probably belong to the Anguillulides and had gained access to the skin of the animal while lying on infested places.

In deer Kless found the *Filaria flexuosa* (*Fil. terebra*) which gives rise to the formation of nodules in the subcutaneous connective tissue: isolated nodules of this kind were visible on the skin as bluish and white elevations.

**Literature.** Cinotti, N. Erc., 1906, 466.—Kless, Z. f. Flhyg., 1908, XVII, 116.—Künnemann, D. t. W., 1905, 269.—Schneider, Ö. M., 1894, 337.—Siedamgrotzky, S. B., 1883, 19.



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